

# *Elements of Surgical Pathology*

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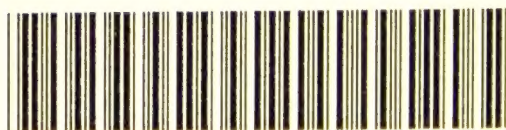
*Augustus J. Pepper M.S., M.B.*

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ELEMENTS  
OF  
SURGICAL PATHOLOGY





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OF  
SURGICAL PATHOLOGY

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ILLUSTRATED WITH 99 ENGRAVINGS

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FOURTH EDITION

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## PREFACE TO FOURTH EDITION.

THE advance in general and surgical pathology since the issue of the Third Edition has necessitated so many changes that the original text has been entirely re-written. "Mycetoma," "Atlo-axoid Disease," "Mastoid Disease," "Myositis Ossificans," "Appendicitis," "Renal Tumours and Calculi," and many other subjects, together with eighteen engravings, have been added to the work.

The author cannot too strongly express his gratitude to Mr. T. H. R. Crowle, F.R.C.S., for the valuable suggestions he has made, and the great assistance he has given generally during the passage of the volume through the press. He desires also to record his obligations to Mr. J. Jackson Clarke, F.R.C.S., for the selection and arrangement of many of the new illustrations.

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# SURGICAL PATHOLOGY.

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## CHAPTER I.

### INFLAMMATION.

**Definition.** -It is difficult to frame a concise definition of inflammation, since the changes involved in the process are so numerous and vary so much in extent in different cases. The definition given by Dr. Sanderson is perhaps the best. It is as follows: "Inflammation is the succession of changes which occurs in a living tissue when it is injured, provided that the injury is not of such a degree as at once to destroy its structure and vitality."

Now it must be understood that no form of injury, whether it be mechanical, thermal, or chemical (*e.g.* a white hot cautery or a concentrated solution of zinc chloride) can be inflicted which shall result in necrosis only; for however intense the cause and however suddenly it acts, there is formed an intermediate zone between the eschar or slough and the uninjured structure, a zone in which inflammation is set up.

It is generally admitted that the primary change is a molecular alteration in the walls of the vessels (Cohnheim, Samuel, Lister), and that this is the cause of the vascular dilatation. The increase in the physico chemical changes going on during the progress of an inflammation is indicative of lowered vital power; for the more rapid the metamorphosis the less is the stability, and so whilst inflammation is

constructive in so far as it leads to an accumulation of indifferent cells, it is destructive as regards its influence on the tissues.

The older views of the essential pathology of inflammation now practically exploded may be briefly summarised: (1) The *neuro-paralytic*; here it was supposed that irritation of the sensory nerves of the part brought about a dilatation of the vessels by inducing a reflex paralysis of the vaso-motor nerves. (2) The *neuro-spastic*, which was grounded on the belief that instead of being paralysed the arteries were stimulated through their nerves, and that, answering to the irritation, they contracted. The hyperæmia was attributed to dilatation of the corresponding capillaries and veins. In refutation of these hypotheses it may be noted that inflammation of the cornea may not show itself for several hours after the part has been irritated, whereas if the injury acted through the nerves it ought to manifest itself much more quickly. It cannot be denied that the calibre of the vessels and the vascular tension are altered by conditions that disturb the functional equilibrium of the vaso-motor nerves, and vary the amount of physiological contraction of the muscular elements. Stricker has shown that certain fibres run in the sciatic nerve, which, on being stimulated, cause a direct dilatation of the vessels by inhibiting the action of the vaso-motor nerves. Again, division of the cervical sympathetic, in the rabbit, for example, is rapidly followed by a flux of blood to the ear, the vessels dilating on account of the paresis of their walls. But these phenomena are mainly, if not entirely, dependent on a departure from the normal state of contraction of the muscular fibre cells by reason of disturbed innervation, and not from a molecular change, the result of damaged nutrition. This molecular change may be taken, then, as the differential sign between simple hyperæmia and

inflammation. (3) The theory which assumed that inflammation was the consequence of an increase in the attraction the tissues were supposed to have for the blood-plasma which nourished them; and that the immediate cause of this was a stimulus acting on the cells, exciting them to greater nutritive and formative activity. Virchow made this hypothesis the basis of his "cellular pathology." One fact, however, is sufficient to negative it, viz. that inflammatory exudation from the vessels may take place even when the connective tissue corpuscles of the part have been killed by the severity of the injury. The regenerative changes in connective-tissue corpuscles described by Senftleben, explain in some measure the discrepancy in the statements concerning the origin of indifferent embryonic cells found in inflamed tissues; what was considered as evidence of primary inflammatory segmentation of cells was probably their secondary growth and proliferation incidental to repair.

**Vascular changes.**—"Vascular disturbance is the most important and the most characteristic factor in the entire process of inflammation." The first alteration noticed is a general dilatation of the vessels, which commences in the arteries, and rapidly spreads to the capillaries and veins; though it is said that in response to some stimuli —*e.g.* ammonia—there is a primary contraction of short duration. The cause of the dilatation, as before stated, is a molecular disturbance in the tissue elements that construct the vessel walls, entailing a loss of muscular excitability and contractility. As the impairment of nutrition increases, the power of restraining the undue passage of the constituents of the blood into the surrounding tissues becomes less; in other words, inflammatory exudation ensues. The capillary blood stream which underwent a short-lived initial acceleration owing to the relatively greater dilatation of the arteries, now shows a marked

diminution in the rate of flow, especially in the veins, whilst "that in the capillaries necessarily participates in it." The slowing culminates in complete stoppage or *stasis*, if the original cause of the inflammation is severe in degree or prolonged in its application. Stasis soon leads to thrombosis. Dr. Sanderson believes that the calibre of the vessels has little or no influence in causing the retardation, since the veins and capillaries are dilated both during the initial acceleration and the subsequent retardation. Lister suggests that changes in the endothelium may cause the slowing of the blood current "by increasing the frictional adhesion between the blood and the vessel wall." The causes of the ultimate arrest of the current are not far to seek; the lumen of the vessels is choked with corpuscles, and the capillaries readily compressible are strangled by the pressure of the accumulated exudation. At this period, too, the back pressure from the veins adds to the existing difficulty in the circulation, and the volume of blood passing from the part in a given time is much diminished. It may be here stated that in one case Cohnheim found the quantity of blood discharged by the local veins was at the *commencement* of the inflammatory process more than double the normal amount.

The vascular tension varies; at first it is diminished "owing to a source of resistance developed in the smallest arterioles and capillaries." This can scarcely be the result of the dilatation, "for the arterioles receive their full supply of blood, and as yet there is no difficulty in escape from the veins." One fact must not be lost sight of: a large quantity of fluid oozes from the walls of the blood-vessels, and is rapidly taken up and carried off by the lymphatics. This is one reason why there is so little swelling in the earlier stages of inflammation; it is only when the exudation gets more corpuscular that it coagulates.



the coagula acting as a barrier to the passage of fluid from the blood-vessels to the lymphatics.

The **lateral pressure** in the blood-vessels is raised later by the causes that impede the delivery of blood by the veins, and exudation by the lymphatics. Among the former may be mentioned thrombosis and extravasation. The denser the tissue affected, the greater is the resistance offered to the expansion of the walls of the vessels, and the greater, consequently, is the blood pressure.

The volume of the exudation, though suggestive, is not conclusive of the intensity of the inflammation; the quality must be taken into account as well, and here one sees the striking difference between mere hyperemia from disturbance in the vaso-motor apparatus and inflammatory engorgement of the vessels. In the former case the exuded fluid is serous; in the latter it is highly albuminous, corpuscular, and coagulable; nevertheless, many of the phenomena of inflammation are in reality exaggerations of normal physiological conditions, for in health the calibre of the vessels is frequently varying, exudation of fluid by osmosis is in constant process, and the white blood corpuscles habitually wander to and from their vascular abodes.

Coincidental with the slowing of the blood current very remarkable changes can be seen within the vessels.

In the normal state a clear narrow channel, filled only with liquor sanguinis, exists between the inner coat of the vessel and the central or axial stream; but as the velocity of the circulation diminishes the blood corpuscles assume a "peripheral disposition," especially in the veins. The clear space above referred to becomes crowded with blood corpuscles, particularly leucocytes, which in the first instance roll slowly by the wall of the vessel, halting now and then

in their onward progress. Soon, however, they show a tendency to fix themselves, and eventually they succeed in doing so. Then commences the process known as *diapedesis*, or migration into the surrounding tissues. How far this is accomplished by their own motility is matter of question. Some pathologists believe that the "peripheral disposition" is purely the result of mechanical action, that the leucocytes, in short, are driven into the slackened side current by reason of retardation of the axial one, instead of wandering thither by their intrinsic movements. The explanation given of the preponderance of white over red corpuscles at the margin of the sluggish blood stream is, that the former are specifically lighter than the latter, and thus are the first to be carried outwards. The balance of evidence seems to be in favour of the theory which supposes them to take an active part in their transportation, and, to say the least, this explanation is in conformity with what is known of the general life-history of leucocytes. Besides, it does not follow that because solid particles can be diffused through a fluid by external agency that they always are so.

**Diapedesis of leucocytes.**—The walls of the capillaries being formed of cells, held together by a soft ground substance, readily allow of the passage of leucocytes through them. These bodies may be seen in different stages of transit, their amoeboid offshoots always being directed from the lumen of the vessel; whether they pass through open spaces, or stomata between the epithelioid cells, or make channels for themselves, is not quite certain.

It was held by Arnold that in the normal state very minute apertures, or *stigmata*, are to be found between the cells, and that they become enlarged into *stomata* during inflammation. Cohnheim and others have demurred to this view, on the grounds that

inflammatory exudation fluid contains more albumin and coagulable material than does liquor sanguinis, whereas if it were simply a case of percolation through open pores it ought to have a similar composition. Moreover, stained preparations show that these supposed stomata are probably altered intercellular substance.

Cornil and Ranvier assert that minute openings through which the leucocytes have passed can be shown by the aid of nitrate of silver, but this view is highly speculative. According to Arnold, they pass out chiefly where the intercellular substance or cement is in greatest quantity. Cohnheim insists that the exudation is dependent upon a molecular change in the filter by which the percolation of fluid and the transportation of corpuscles is facilitated.

It certainly is not due to the retardation of the blood stream, for mere slowing is not followed by extravasation of cellular elements. Perhaps the strongest evidence in support of Cohnheim's view is, that if the blood supply to a part be entirely cut off, say for twenty-four hours, by the application of a ligature, and afterwards re-established, the vessels will not only dilate, but exudation will take place, and a veritable inflammation ensue.

Having cleared the vessels, the leucocytes wander into the surrounding tissues, and collect in groups around the fixed corpuscles. It was this aggregation that led Virchow to believe that the small groups of cells were derived from segmentation of connective-tissue corpuscles.

The migration of leucocytes takes place from *veins and capillaries*. Red blood corpuscles traverse the walls of the *capillaries*, but not by amoeboid movements. They are driven through by the intravascular pressure, the molecular change in the walls of the vessels and the previous passage of leucocytes

favouring this transportation. Probably neither white nor red corpuscles escape from the arteries.

It has already been said that the fluid portion of the exudation is highly coagulable, and that it coagulates outside the vessels under the influence of the white blood corpuscles. Though it is customary to speak of "fibrinous exudation," it must be remembered that fibrin, as such, does not exude from the vessels.

The elements of coagulation are supplied as follows: The *fibrinogen* by the fluid portion of the exudation, and the *fibrin ferment* and *fibrinoplastin* by the disintegrating white corpuscles.

The coagulated portion of the exudation is known as "inflammatory lymph."

In the exudation is a quantity of *mucin*. This is probably derived from a mucoid transformation of the protoplasm of the cells. It is best seen in discharges from inflamed mucous membranes, in the glairy secretion from some ulcers, and in the layer of embryonic tissue on the surface of articular cartilage in white swelling. There are important changes in the tissues themselves; the intercellular matrix, whether fibrillated or homogeneous, liquefies from mucoid softening (*Rindfleisch*), a result probably due to the quasi-digestive action of the white blood corpuscles. The epithelioid cells of the blood-vessels swell, and help to obstruct the lumen; and thus, aided by the pressure from without, and the adhesion of leucocytes to the interior of the vessels, thrombosis ensues. Even yet resolution may happen, but a step farther takes us to actual destruction of tissue by suppuration or gangrene.

Scrapings from an acutely inflamed tissue contain cells of different size and form:

(a) Cells with a single nucleus.

(b) Pus cells in which the nuclei, varying from two to five, are brought into relief by the action of

acetic acid, which clears up the albuminoid particles that render the cells granular.

(c) The so-called compound inflammatory corpuscles of Gluge are only ordinary cells, enlarged and loaded with fat molecules; they are most plentiful in tissues rich in fatty matter, and hence are seen in numbers in softening of the brain and spinal cord.

Filaments of fibrin are also seen enclosing the cells in their meshes, and if the specimen has been hardened, coagulated strings of mucin are present.

The majority of the cells are undoubtedly migratory corpuscles that have escaped from the blood-vessels; some are derived from endogenous formation and subsequent dissociation, and a few, perhaps, are obtained by gemmation and fission of multinucleated masses of protoplasm. Cohnheim supports the doctrine of migration pure and simple; Cornil and Ranvier maintain that segmentation of connective tissue corpuscles is an important factor in the production of cells.

There seem at any rate sufficient grounds for refusing to admit that the blood furnishes all the indifferent cells found in inflamed tissues. In the case of cartilage Redfern has shown that the cells may be seen in different stages of growth and proliferation; the nuclei enlarge, and the protoplasm swells up, division ensues, and the capsules being dissolved, the broods are set free, and joining adjacent groups, a layer of embryonic tissue is formed on the surface. In bone the corpuscles appear perfectly passive (Billroth), even when the lucunæ are opened up by absorption.

Stricker, whilst admitting that in inflammation of the cornea and areolar tissue most of the cells have wandered from the vessels, alleges that segmentation of the fixed corpuscles can be made out on careful observation,

It should be stated, however, that the latest writers on the pathology of inflammation have almost unanimously rejected the theory that the fixed connective corpuscles take part in the production of inflammatory cells.

Concerning the epidermis and mucous epithelium, there can be little doubt that cell proliferation is very active, for apart from the results of investigation into the anatomy of inflammation, there is the fact that in these tissues a continued physiological succession of cells goes on, new elements supplanting those whose term of life and function has expired. It may be stated generally, however, that diapedesis of leucocytes accounts for by far the greater number of inflammatory cells, and that the segmentation of corpuscles is in inverse ratio to the severity of the inflammatory process. At the same time, if the vascular changes are very slight, the tissues outside the blood-vessels may escape altogether.

Many pathologists, whilst contending "that injury only kills or impairs the vitality of the tissues beyond the blood-vessels, that it never induces multiplication of cells from excitation from without," admit that the fixed corpuscles proliferate when the cause of the inflammation is removed, and that this proliferation is a purely regenerative one, and is induced by "the changes in the environment of the cells, the result of the inflammation."

**Signs of inflammation.**—Redness, pain, heat, and swelling, the four stereotyped signs of inflammation, may severally, or in combination, be met with in other conditions. Thus redness, pain, and heat, may be observed in joints, the seat of nervous mimicry of disease (Paget); swelling from exudation, as in transient erythema, and pain so momentary as not to admit of inflammation as a cause. In inflammation itself these signs are associated in different degrees;



thus, a joint may be filled with pus in purulent infection, with but little redness of the synovial membrane, or it may be intensely congested in rheumatism with only a slight amount of exudation.

The *redness* is mainly due to dilatation of the vessels. Exudation of red corpuscles shares in its production, and in some diseases there is considerable discharge of the hæmoglobin from the corpuscles, which, passing out in a state of solution, stains the bodies with which it comes in contact; *e.g.* the brick-dust colour of the lymph in syphilitic iritis. Lastly, in long-standing hyperæmia the blood pigment is taken up and fixed by the cells.

The *pain* is caused chiefly by the tension on the filaments of the nerves from the pressure of the exudation, but partly by a chemical irritation of the inflammatory products acting upon the ends of the nerves; for the pain is not always proportionate to the extent of the stretching.

The *local heat* depends upon the increased amount of oxygenated blood, and the active chemical changes going on in the part. The *swelling* is the result of the hyperæmia and exudation.

Inflammation causes an alteration in the function of tissues, and this may be quantitative or qualitative, or both, *e.g.* the secretion from an inflamed salivary gland is changed in amount and character. Muscles are more or less paralysed, as seen in the heart affected with myocarditis. This direct inflammatory paralysis must not be confounded with the reflex inhibition of muscular action, consequent upon inflammation of adjacent structures.

**Ætiology of inflammation.**—There are many different causes of inflammation, but the proximate one in all cases is something that induces a molecular disturbance in the walls of the blood-vessels. The injury, whether it be mechanical, thermal, or chemical,

must not be so severe as to kill the tissue outright. Now, although it cannot be denied that the above-mentioned modes of violence are in themselves sufficient to excite inflammation, "it is now regarded as certain by pathologists that minute organisms are concerned in all the more serious pathological effects of injuries" (Sanderson). This is proved by direct and indirect evidence. In the first place, tissues may be spoiled and their functions annihilated without any commensurate inflammatory change being set up, provided the access of septic germs to the injured tissue be prevented. Hueter, in a series of antiseptic operations, showed that he could kill large portions of tissue without, as he thought, exciting inflammation. In one of these experiments he incised the skin, thrust a white-hot cautery into the muscle beneath, and then closed the wound, with the result that it healed by first intention. The inflammatory reaction was no doubt very slight, but from what is known about the effects of injuries generally one can scarcely think it was entirely absent; moreover, Hueter's interpretation of the results of his investigations must be read by the light of another of his views; viz. that the callus formed about a fracture is not an inflammatory product, but the material elaborated for the process of simple physiological repair of an injury. At the same time, he does not deny that "emigrated leucocytes are concerned in the formation of callus."

Again, he says, "true inflammation is characterised by the tendency to suppuration," apparently excluding those cases which, though transient, are yet of the same nature as the more severe forms. So strongly is Hueter imbued with the belief that germs are the essential cause of inflammation, that he remarks, "inflammation is an epidemic which is spread over the whole world." The author, whilst considering this theory too exclusive, freely admits that



organisms play an important part in the causation of severe cases of inflammation both as regards local destruction of tissue and remote infective processes.

Chauveau's experiments on the spermatic cord are very instructive. This pathologist, after freeing the testicle from the dartos, twisted the cord to such an extent that the patency of the spermatic vessels was destroyed. The result was that the testicle underwent atrophy and absorption (in the same way as a simple or non-infective embolic infarct, *q.v.*) without causing inflammation in the living tissues around. He then varied the procedure by first injecting into the general circulation the fluid portion of pus derived from a septic abscess. This meant that organisms were present in the testicle at the time the circulation in the gland was arrested. After a while the part became swollen and inflamed, and suppuration was established outside the organ, which in the meantime had become the seat of putrefactive decomposition.

With regard to the so-called *cryptogenetic* inflammations, *e.g.* ulcerative endocarditis and infective osteomyelitis, or those in which the cause of the acute pathological process is, or rather was, obscure, it may be said that recent observations have shown that they are dependent on micrococci which have gained access to the circulation by one or other of the natural passages of the body, *e.g.* the alimentary tract. In these diseases it may be said that the local products of inflammation are crowded with organisms, and that too, without there having been any direct exposure to the atmosphere. There is every reason to believe that the predisposing cause of these inflammations is an impaired vitality of the tissues doomed to die from an acute infective process. Ulcerative endocarditis is always the sequel of degeneration of the cardiac valves from previous inflammation. Acute osteomyelitis and periostitis can be shown experimentally to be largely

dependent on the presence of germs; *e.g.* if a bone be simply injured mechanically, the inflammatory reaction rarely ends in local suppuration and general pyæmia, whereas if, prior to the infliction of the injury, septic fluid containing organisms be injected into the veins, this termination is very common. It may reasonably be supposed that in spontaneous or idiopathic osteomyelitis there is a pre-existing textural change in the bone which disposes it to destructive inflammation. From the foregoing statements the following conclusions may be drawn: (1) Anything which lowers the vitality of a tissue renders it more liable to succumb to the exciting causes of inflammation. (2) Those conditions which increase or depress the natural excitability of a tissue, if pushed far enough, end in its death, or short of this in inflammation. (3) Whilst physical violence such as heat, cold, mechanical injury, and chemical action undoubtedly act as exciters of inflammation, the result is much more serious if the injured tissue is at the same time exposed to the influence of infective organisms.

**Modes of termination.** -Inflammation may terminate in resolution or absorption, organisation or new formation, or in death of the tissue by suppuration, gangrene, or caseation.

**Resolution.** Some of the cells re-enter the blood-vessels, others undergo molecular disintegration and absorption by the lymphatics, whilst a few may remain as fixed connective-tissue corpuscles (fibro-plasts). The fibrin becomes granular, disintegrates, and is then removed. The same change occurs in any capillary thrombi that have formed. The endothelial cells of the capillaries shrink to their former size, or if degenerated past repair, are replaced by others. The vitality of the contractile walls of the capillaries and arterioles is re-established, and the vessels assume their proper calibre; "the blood brings material for

regeneration, which is used when the cause of the inflammation is gone." It may be here remarked that the expression "venous absorption" is often used erroneously; of course the walls of veins are permeable to fluids, and leucocytes traverse them; but still the chief part of absorption is capillary, be it blood, vascular, or lymphatic.

It must not be supposed that the tissues have necessarily regained their normal condition when all the more obvious signs of inflammation have disappeared. The inflammatory process has been described as consisting essentially of a molecular disturbance of the walls of the vessels, to which are added exudation, and those alterations of the extravascular tissues consequent on disturbed nutrition. Now it should be borne in mind that for some time after the exudation has been removed by absorption, there is a molecular instability of the tissues which renders them more than usually prone to react to the exciting causes of inflammation. The loss of tone, though latent so long as the parts are kept at rest, soon becomes manifest when they are subjected to strain or other form of irritation, it may be only to the extent of normally healthy exercise. The truth of this is nowhere better exemplified than in the case of an injury to a joint. An attack of simple serous synovitis of the knee *e.g.* may have to all appearances subsided, but the surgeon knows that unless physiological rest is continued after the apparent need for it has been removed, there will not improbably be a renewal of the inflammatory disturbance.

The modifications of the life processes in tissues due to inflammation are more varied when the cause of the latter is some poison charged with living organisms. Then, although the tissues, as in cases of simple traumatic inflammation, are weakened in their power of withstanding injury, they may be strengthened in

their antagonism to the germs of disease, or in other words, they may have received a greater or less immunity from subsequent attacks.

Resolution takes place more quickly and perfectly in the tissues of the young, (1) because in them there is less chance of hindrance from acquired defects of nutrition ; and (2) in children there is a more rapid substitution of formative material for that which has been rendered waste in the work of development.

**Organisation.**—Should the inflammation be prolonged and moderate in degree, organisation takes place in the exudation without visible loss of tissue. New blood-vessels are formed by a looping of the old ones, the softened walls of the capillaries allowing protrusion here and there. The channels of contiguous offshoots merge by absorption of the intervening walls, and so a vascular network is established. There is, besides, a separate development of new vessels in the inflammatory embryonic tissue. This may be effected in two ways: (1) by the loosening and disintegration of the central cells of columns, whilst the peripheral elongate to construct the walls of capillaries; (2) vaso-formative cells send out canalicular processes which anastomose. The cells expand, the protoplasm liquefies, and the nuclei possibly grow into the first blood corpuscles. Be this as it may, these newly-built capillaries join the pre-existing vessels. Exactly the same changes are observed in the growth of tumours, such as sarcomata, the type of which is embryonic connective tissue.

This vascularised lymph constitutes **granulation tissue**, which is merely a mass of indifferent cells, cemented by a scanty amount of ground substance, and traversed by capillary blood-vessels. It is termed granulation tissue, because when formed on an inflamed surface, the centrifugal pressure directs the loops of vessels in the path of least resistance, and the

exudation corpuscles arrange themselves about the loops. The old tissue disappears before this inflammatory neoplasia, which, when the irritation ceases, organises into connective tissue.

On the subsidence of the inflammation the granulation cells proliferate with a view to repair. They enlarge and multiply by division, and their protoplasm fibrillates, hence they are called "fibro-plasts." The fibrils of contiguous cells by joining one another form strands of fibrous tissue. Multinucleated cells are usually to be seen; they originate in two ways: (1) by division of the nucleus without that of the protoplasm; (2) by the fusion of uninucleated or binucleated cells.

The new formed tissue contracts, thus obliterating many of the vessels, so that *a scar*, which is at first more vascular than the surrounding tissue, becomes smaller and paler with the lapse of time, and eventually (unless pigmented) quite white. As a rule it is depressed; but if the irritation be prolonged, as sometimes happens in the case of burns, the cicatricial tissue is so abundant as to project above the surface as a keloid mass.

There is always a tendency during organisation to reproduce the original tissue of the part, as we shall see when treating of the mode of union of wounds. The more highly developed the tissue, the less is this tendency developed; thus, in the human subject, at least, the ends of divided muscles are mainly cemented by fibrous tissue, and cicatrices of the skin do not contain sebaceous or sweat glands.

**Suppuration.**—If this takes place with loss of tissue on the surface, the process is described as ulceration; if in the substance of tissues or organs, an abscess is the result. We have only to suppose an intensified irritation to see that not only will the exudation of leucocytes and liquor sanguinis be

increased, but the liquefaction of the cells and intercellular substance will be complete. Moreover, there will be extensive occlusion of vessels by pressure from without and coagulation within. The tissues suffering acute starvation quickly disintegrate. These changes are more rapid if the inflammation be

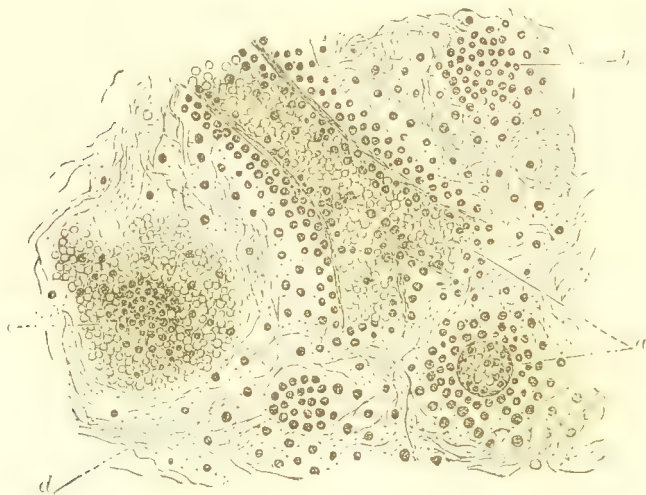


Fig. 1.—Suppurative Inflammation of the Cerebrum ( $\times 250$ ).

The section was made half an inch away from the situation of numerous viscid abscesses: *a*, blood-vessels, showing leucocytes collected within and without the walls; *b*, "microscopical abscess;" *c*, the same in the midst of a capillary extravasation; *d*, brain substance, showing delicate fibres and granules, the result of inflammatory softening and fatty degeneration. The normal histological characters have disappeared.

infective, for then molecular destruction results from the influence of minute organisms.

Here we have the mode of formation of an **acute abscess**, *i.e.* a closed cavity filled with the debris of broken-down tissue, and fluid and cells derived from the vessels. This is the final stage of an acute inflammation. The wall of the abscess consists of hyperemic disintegrating tissue, the so-called *pyogenic membrane*, but it contains no vessels



of new formation. From the vessels of this layer exudation passes into the cavity. Add to this a continuous melting away of the boundary wall itself as the inflammation spreads, and the picture of an enlarging abscess is complete. It may be noted that suppuration does not start from one focus, for under the microscope numerous minute centres of pus formation may be observed (Fig. 1). These microscopical abscesses increase in size, and coalesce.

In addition to the original cause of the inflammation there is now the added tension from the retained products of suppuration. The tension is removed by the bursting of the abscess. The importance of early relief from the pressure, in order to check the destruction of tissue and the absorption of intoxicating or septic material, is obvious.

Blood-vessels and nerves resist the action of these inflammatory changes longer than other tissues, except tendons and the calcified framework of bone.

When an acute abscess has been opened its walls collapse, the opposed surfaces adhere more or less, and the suppurating area is greatly diminished.

From the opening in the abscess a thick creamy fluid escapes, mixed with blood from ruptured and divided vessels. The yellowish-white colour is due to the suspension of refractive bodies (pus cells) in a fluid termed *liquor puris*. To see that this is so, we have only to allow the pus to stand for a time in a glass vessel, when two distinct layers will be observed, the lower a yellowish-white deposit with uniform surface, the upper a clear fluid. Chemical analysis proves this fluid to be practically identical with *liquor sanguinis*, which is no doubt its source.

If some of the lower layer be examined with the microscope, it will be found to consist mainly of corpuscles from  $\frac{1}{2500}$ th to  $\frac{1}{3500}$ th of an inch in diameter. These bodies are for the most part round,

but some are crenated or otherwise misshapen. No boundary wall can be seen. They look cloudy or granular, from minute fatty and albuminoid particles. Acetic acid causes the cells to swell up and become clear. Many of the older cells are coarsely granular from fat molecules which are soluble in ether. The fat is chiefly derived from degeneration of the protoplasm of the cells, but some, probably, has been taken up from the debris of perished tissue. The so-called compound inflammatory corpuscles of Gluge are of this nature. Pus cells occasionally contain pigment. *Blue pus* owes its colour to the presence of an organism — micrococcus cyaneus. There are also numberless free granules, the product of disintegration of the cells. Simple or *laudable* pus is alkaline in reaction. It forms a viscid gelatinous mass on the addition of liquor potassæ.

**Origin of pus corpuscles.**—The majority are leucocytes that have migrated from the blood-vessels, but their number is increased in the discharges from mucous membranes, and the exudation from serous surfaces. In the two latter situations there is a constant physiological reproduction of cells.\*

They resemble white blood corpuscles, in that whilst living they possess the property of spontaneous movement. Their vitality, however, is low, and they soon become motionless and degenerate. The fact of their containing two or more nuclei is, perhaps, a sign of declining nutritive activity.

According to Ogston, the pus taken from all acute abscesses is charged with micrococci.

**Gangrene.**—Inflammation is the most common cause of gangrene or death of visible portions of tissue. The blood current is obstructed by the pressure of the exudation, and the vessels are blocked

\* For modes of cell multiplication *vide* page 106.



by coagula; hence the tissues perish from acute starvation before there is time for complete disintegration of their individual elements. In the necrosed masses the original structure is recognisable.

**Caseation.**—In chronic inflammations, especially of the lungs, bone, and lymphatic glands, the exudation together with the affected tissue undergoes fatty degeneration and disintegration, the more liquid portion is absorbed, and a putty-like substance remains; this consists chiefly of granular debris, in which crystals of stearic acid and plates of cholesterine are formed. The process often terminates in calcification.

It may be remarked that the discharge from *gouty abscesses* is loaded with what is often erroneously spoken of as chalky matter, but which in reality is urate of soda in the form of acicular crystals, scattered or in stellate groups.

**Chronic inflammation** is the result of long-continued local irritation, or of some constitutional weakness. The inherent defect in nutrition may be general, or centred in certain organs or tissues; *e.g.* in tubercular disease the bones, joints, lymphatic glands, and the lungs suffer most.

The predisposing condition is called a *diathesis*. It was believed by the humoral pathologists that a special *materies morbi* existed in the blood, and that this was the cause of the local disorder; hence the expression “cancerous *deposit*,” malignant “growths” being regarded as exudations of *cacoplastic lymph*.

On the other hand, the solidists laid the foundation of all disease in the tissues themselves, considering the blood as being secondarily affected. We know, however, that it is a “flesh and blood” malady, for it is difficult to conceive how one can be affected without the other. It is true the specific nature of a blood poison may disappear, as shown by the failure of inoculation, and by the non-transmission of syphilis

in the tertiary stage from parents to offspring; (and yet how often do very grave inflammatory lesions crop up in after years). Some pathologists look upon these as sequelæ of syphilis, and, in so far as a reproduction of the same disease in others is concerned, they may be; yet it cannot be supposed that the blood is free from taint.

The final result of a chronic inflammation turns mainly upon whether the latter proceeds from a local or constitutional cause. If purely local and only moderately severe, the lymph organises into connective tissue pure and simple, or with the admixture of a more highly-developed product, such as bone.

In tuberculosis caseation and abscess are very common; *e.g.* in the cervical glands.

**Chronic abscess** is also called *congestive*, from the presence of passive rather than acute hyperæmia, and *cold*, from the fact that there is little or no elevation of temperature of the part, for the blood current is sluggish and the chemical changes are not very active.

It is often *consecutive*, as when cervical glandular abscess follows caries of a tooth, or psoas abscess depends upon disease of the spine. From the persistence of the original cause, together with the continued tension, chronic abscesses frequently attain a great size; and when deeply-seated and the resistance of the tissues around is unequal, they follow well known anatomical paths; thus, a spinal abscess may open at the groin or knee, or even at the ankle. And, inasmuch as the line of least obstruction is usually in the long axis of the body, they *gravitate* to more dependent parts. The structures around become condensed. The cicatricial tissue, as it shrinks, diminishes its own vascularity, and makes the walls of the abscess rigid, so that they can only imperfectly collapse when the cavity is laid open. As the contents escape, germ-

laden air too often enters and leads to decomposition. The badly-nourished walls are slow in constructing granulations for the obliteration of the space within, and are apt to absorb infective matter, the source of chronic and acute pyæmia. Hence the force of Billroth's remark, when speaking of psoas abscesses, "Be thankful for every day they remain closed." But the present state of perfection of antiseptic surgery not only renders the opening of chronic abscesses admissible, but generally advisable, and for several reasons:—(1) They may be on the verge of bursting, in which event the openings would be insufficient for proper drainage. (2) They may be encroaching on some important organ or structure, *e.g.* retropharyngeal abscess. (3) The probability of their opening later in a less advantageous position. Moreover, chronic abscesses are often of tubercular origin, and it is then desirable to get rid of their contents to prevent secondary infection of other parts. The contents of chronic abscesses vary much. The pus may differ but little from that found in an acute abscess; it may be thin and watery ichorous—containing flakes or curds of lymph; and, lastly, by absorption of the greater portion of the liquor puris it may be reduced to the consistence of clotted cream or putty, when it is designated "inspissated pus."

When the abscess has formed in connection with diseased bone, the pus contains an excess of lime salts, and not unfrequently minute sequestra.

Abscesses arising at the seat of a previous inflammation are termed "residual" (Paget). The vitality of the tissues has remained defective, and unable to cope with further irritation. The part is a "*locus resistentiæ minoris*."

## CHAPTER II.

## PAIN.

PAIN is always a symptom of diminished functional and, therefore, nutritive power, whether it be the headache from a tired brain or the smarting from a scald. We say functional *power*; for excessive functional *activity* is one of the first indications of exhaustion (C. Bernard).

It is purely subjective, and is, therefore, difficult to estimate; for its existence, nature, and extent can be simulated, and it may appear out of all proportion to the intensity of any assignable cause. It is the fashion to give but little heed to the sufferings of hysterical patients, but their nervous mimicry of pain is to them a real disease.

There are three factors in the production of pain; (1) Extrinsic irritation, physical or chemical; (2) inherent susceptibility of the implicated nerve-centre; (3) emotion. It is well to assume that all pain is organic (it is termed functional when no corresponding tissue-change can be seen, but "cell" and "fibre" are only coarse expressions of molecular structure), for then there will be no excuse for considering the less obvious cases as "idiopathic." The irritation may be of peripheral or central origin. When central, it is referred to the whole or some part of the area of distribution of the corresponding nerve. When peripheral, it is usually referred to the seat of irritation, but not rarely to the terminal part of some other branch of the same nerve, as in the knee when the hip is diseased, or in the ear when a tooth pulp is inflamed; or the pain is felt in some nerve associated in function, origin, or distribution, as when

ascarides in the rectum leads to discomfort about the penis, or a stone in the bladder causes painful tenesmus. If the stimulus be applied in the course of a nerve trunk, as in neuritis, there is local tenderness to the touch, accompanied by pains radiating along the derivative branches. In some cases the pain is experienced only at the distal end; *e.g.* at the back of the head in atlo-axoid disease, in which the great occipital nerve is caught as it passes between the bones.

In any case of localised pain the nerve should be examined as far back to its origin as practicable, and then the trunk and all its branches taken in review.

The localisation of pain in neuralgia has received an anatomical explanation from the observation made by Mr. Horsley, that true "nervi nervorum" exist in nerve trunks. They run as single medullated fibres in the connective tissue of the epineurium, and terminate in end bulbs very similar to those found in the conjunctiva.

**Types of pain.**—There are two primary types of pain: (1) continuous; (2) intermittent. The former is either *aching*, which is symptomatic of tension beneath very resisting structures, as in otitis, periostitis, and subfascial inflammation, and in the erosion of vertebræ by an aneurism; in all these instances the nerves are constantly on the stretch; or it is *smarting*, which is characteristic of surface irritation, as in burns and scalds. (The latter is in reality an intermittent pain, the impulses succeeding one another so rapidly as to make it appear continuous, the analogy being that of tonic to clonic spasm.)

Intermittent pain is either *neuralgic* or *throbbing*. In the former case it is indicative of recurring discharges of a diseased nerve-centre as in the "lightning pains" of locomotor ataxia; or of varying tension, as when the pulp of a tooth is inflamed.

In throbbing pain, the nerve endings are oversensitive from inflammation, and the tissues are softened, and so allow considerable latitude for expansion of the vessels; hence the pulsation of the arteries is felt by the patient, and this gives the peculiar character to the pain. It is suggestive of the formation of pus. The *shooting* pains felt in cancer are probably due to the implication of fresh nerve-fibres by the growth. A combination of continuous and spasmodic pain is well seen in the passage of a biliary or renal calculus, the continuous character depending upon the resisting nature of the walls of the duct and the spasmodic upon the intermittent contractions of the muscular coat.

**Effects of pain on nutrition.**—There can be no doubt but that nutritive changes are more marked where pain is severe, the atrophy of the muscles in hip-joint disease being proportionate (*ceteris paribus*) to the patient's suffering; for local, like general, disturbance of rest prevents the natural repair of tissues wasted during activity.

**Reflex subdual of pain.**—As pain can be induced by reflex action, so it may be subdued, hence the use of powerful local sedatives applied to the skin; *e.g.* in the deep-seated inflammation of joints. The relief given by counter-irritants, though apparently contradicting this theory, really supports it, for the peripheral irritation produces an exhaustion of the nerve-centre, and makes it less sensitive to a stimulus applied to the deep-seated nerves.



## CHAPTER III.

### ULCERATION.

ULCERATION is a surface solution of continuity from molecular death of the tissues. In this way it differs from gangrene or molar death. (Molecular disintegration is not limited to ulceration, for it is met with in primary interstitial degeneration.)

Whatever the primary cause of ulceration there is at some period inflammation of the part; thus, in *cancrum oris* the most important factor is progressive capillary thrombosis, but the coagula irritate the walls of the vessels and cause them to inflame, and the inflammation spreads to the tissues around. An ulcer may begin as a surface lesion, or be the consequence of suppuration beneath the skin or a mucous membrane. An abscess may be defined as a closed ulcer.

**Causes of ulceration.**—The irritation is either physical or chemical, or both; in any case the result is vascular dilatation and exudation, and the tension thus increased acts as a further cause of inflammation, by obstructing the flow of blood and stretching the tissues. As physical causes we may mention wounds, continued pressure and friction, as in bed sores, extreme heat and cold, and rupture of a varicose vein. Chemical irritants may be applied locally to the surface or beneath the skin. The irritant may be the product of specific germs which attack the most vulnerable tissues and cause ulceration, as seen in syphilis and typhoid fever.

**Mode of formation of a simple ulcer.**—We will take the case of the heel pinched by a shoe. In the first place the friction causes dilatation of the vessels and exudation of liquor sanguinis and leucocytes;

hence the redness and swelling. The exudation increases to such an extent that the tissues can no longer retain it within their interstices, and some, escaping between the cells of the softened epidermis, oozes from the surface, as in eczema. If it be more rapid, the epidermis is raised in the form of a bleb. In the meantime the cells of the rete are being excited to greater formative activity. They divide and subdivide, but no longer undergo cornification. The embryonic corpuscles thus derived mingle with the discharge from the vessels. The epidermis becomes soddened, and is swept away by the outward current, or is brushed off by the slightest friction. In this way the tips of the papillæ become exposed. The papillæ themselves are rendered succulent by the exudation and mucoid softening of their constituent elements. This, together with the cellular infiltration, causes them to lose their natural histological features. The exudation now flows from the surface in the form of *pus*. The ulcer is fully formed. The extent of the changes in the subcutaneous tissue varies as the severity of the inflammation.

**The stages of an ulcer.**—There are two opposite processes in the history of an ulcer, destruction and repair. These are so intimately connected that it is difficult to say where one ends and the other begins. But, during the time that elapses from the commencement to the close, an ulcer may be said to pass through three stages: (1) *spreading*; (2) *stationary*; (3) *healing*. These are sometimes erroneously described as varieties of ulcers, whereas the terms really refer to the condition of the ulcer at a given time.

**Spreading period.**—This is well exemplified in a soft chancre. The same causes that led to the formation of the ulcer are still in active progress. The *margin* is surrounded by a zone of hyperæmia, is more or less swollen, is sharply defined and steep.



and, if the ulceration be spreading more rapidly in the subcutaneous tissue than in the skin, it is undermined. As to the *base*, there will be no granulations in the floor of the ulcer, for the inflammatory neoplasia is destroyed too quickly to allow of its assuming the granular form. It has a grey, or yellowish-grey appearance, and is covered with pus and the débris of the disintegrating tissues. The colour is due to fatty matter and softened blood clot, for thrombosis precedes the loss of vitality and subsequent dissolution; and this is the reason why hæmorrhage is not more common. If the ulceration be very active, the cellular tissue is destroyed before the more resisting structures, such as vessels and nerves, have had time to melt away into molecular débris. The base then appears somewhat flocculent and the margin fringed; in fact, gangrene is added to true ulceration. There is usually considerable pain in the part.

**Stationary period.**—This is best observed in chronic varicose ulcers of the leg, whose margins are indurated by condensation of the exudation. This always takes place when the healing is long deferred. The *margin* is usually thickened and rounded. It is rarely so precipitous or undermined as when the ulcer is spreading, nor is the surrounding inflammatory redness so marked. The *base* consists of small, unhealthy, or pale exuberant granulations. The discharge is subject to much variation as to character and amount; at times it is scanty and highly fibrinous, and coagulates on the surface; or, again, it is thin and serous; or these conditions may alternate.

The *causes of ulcers being stationary* are (1) obstruction to the return of blood; *e.g.* where the veins are varicosed (the granulations are then often large and œdematous); (2) continued slight irritation, as in an issue; (3) fixation of the margin to some

rigid underlying structure, which prevents cicatrisation, as in ulcers over the malleoli and crest of the tibia, and over tendons where there is movement in addition.

**Healing period.**—In ordinary circumstances, when the source of irritation has been removed the ulcer begins to heal. In the first place the inflammation tends to resolution, the blood-vessels contract, there is less exudation from the surface of the sore, and that effused into the interstices of the tissues is re-absorbed. The *margin* is fairly on a level with, or slopes towards, the base. Two zones may be distinguished; an outer, where the epidermis is heaped up, opaque, white, and soddened; and an inner, more transparent, where the epithelial cells are only one or two deep, on the surface of the granulation tissue. It has a faintly glistening purplish hue. There is a gradual transition from one zone to the other. The *base* is made up of bright florid granulations discharging pus.

**Histology of the granulations.**—The granulations consist of migratory leucocytes held together by a scanty fibrinous intercellular substance. The deeper cells have greater cohesion than the more superficial ones. Those immediately on the surface are floated off by the fluid that bathes it, and are then known as pus corpuscles. New capillary blood-vessels traverse the cellular mass. They form over-arching loops with the convexity towards the surface. This arrangement is based upon the plan laid down in healthy skin and mucous membrane. It becomes more marked in the granulations of an ulcer, on account of the pressure of the outflowing stream of exudation and the diminished resistance of the softened tissues. Granulations contain no lymphatics, and no nerves of new formation.

**Cicatrisation of an ulcer.**—This is quite as essential to the healing as is the precedent growth

of granulations. Many of the cells undergo fatty degeneration, and are absorbed; some probably return to the blood-vessels by their amoeboid movements; the remainder develop into fixed connective-tissue corpuscles. As the inflammation subsides the deeper cells elongate and become fusiform, and the intercellular substance increases and fibrillates. This highly vascular and corpuscular connective tissue at length contracts, and in so doing obliterates many of the vessels; nor does it stop until the scar is denser and whiter than the normal tissue around. Several months elapse ere the process is completed. As the ulcer heals the epithelial cells grow inwards over the granulations. During this time the contraction may be so great as to cause serious deformity, especially where wide tracts of skin have been destroyed, as in extensive burns. The chin may be drawn down and fixed to the sternum. Ectropion or eversion of the eyelid often follows the cicatrization of an ulcer of the face. In annular ulcer of the leg, there being no skin to glide in the circumference of the limb, the effect of contraction in this direction is to keep up continued irritation of the granulation tissue; hence this form of ulcer rarely heals.

**Reproduction of epithelium.**—As the ulcer heals the epithelium grows over the granulations. The cells are derived from segmentation of those at the margin. Now and then islets spring up at a distance from the marginal zone of epithelium; in these cases the cells of the rete must have escaped destruction by the ulceration, for there is nothing to show that they arise spontaneously from the granulations except under the immediate influence of pre-existing epithelium.

**Reverdin's skin grafting.** All that is necessary is to transplant snippings from the deep protoplasmic cells of the rete to the healthy granulations,

and to fix and protect them there. This, by furnishing fresh centres of epidermal formation, greatly hastens the healing, and by so doing lessens the after-contraction. The grafts are sufficiently fixed, after a few days, to hold of themselves.

**Nomenclature.**—The ordinary features of an ulcer may be so modified as to give to the condition a special appellation. This may arise from a local hindrance to healing, or depend upon some constitutional taint ; or both causes may act at the same time.

**Symptomatic ulcers** are those that point to a disease of wider distribution than the part immediately under observation, such as syphilis, tubercle, venous varicosity, etc. In describing ulcers it is usual to direct attention to the base, the edge, the tissues around, including the vessels that supply and return the blood, and lastly the discharge. The particular designation of an ulcer is drawn from the state of the part that shows the widest departure from the normal ; hence we speak of “diseases of the granulations,” of an “undermined” or “indurated margin,” and so forth. But observation that is limited to any one of these is too exclusive, for it cannot be said that an ulcer is healthy as regards the edge, and not so in respect of the base ; the whole should be passed in review.

**Locality of ulcers.** —There are three factors that determine this : (1) Exposure to injury, whether it be from direct violence or continued irritation ; (2) the local effect of a materies morbi, either at the seat of inoculation, or in some remote part, or both ; thus syphilis leads to primary, secondary, and tertiary ulceration. Typhoid fever attacks the intestine, and sometimes the throat ; scarlet fever and diphtheria the throat ; and small-pox the skin ; (3) inherent or acquired weakness of certain tissues, or, as it is termed, the liability or predisposition to this or that

form<sup>2</sup> of ulceration ; *e.g.* lupus is very prone to occur in the face, and tubercular disease in the small intestine. Varicose ulcers are practically confined to the lower extremities.

Bearing these facts in mind, we have, apart from the characters of the ulcers, a valuable aid to regional diagnosis ; thus the majority of ulcers of the *leg* are either varicose, traumatic, or syphilitic. The widely destructive ulcers of the *face* are due to lupus, syphilis, and epithelioma, which includes rodent ulcer. In the mouth and rectum, malignant disease, syphilis, and local irritation take the lead. In the *small intestine*, typhoid fever and tuberculosis head the list. In the case of the *penis*, the inquiry is very limited after excluding chancres and epithelioma.

Again, in the leg most ulcers begin on the *front aspect*, and this is notably the case in the traumatic and varicose varieties. Varicose ulcers are nearly always found in the lower third ; syphilitic frequently about the knee ; whilst tubercular are more confined to the skin over the epiphyses and about the foot, since these parts are very subject to caries.

**Diseases of the granulations.**—1. Croupous ; 2. Fungous ; 3. Hæmorrhagic ; 4. Diphtheritic.

1. **Croupous disease.**—The only way in which the condition resembles croup is in the existence of a rind covering the base of the ulcer. This may be likened to a false membrane, for it can be peeled off with the forceps. Its presence is compatible with good general health, and it is not contagious. It has a bright yellow or greyish yellow colour. Under the microscope it is seen to be composed of indifferent cells firmly held together by a highly fibrinous material. The formation of this probably depends upon a purely local cause. There is an alteration in the nutrition of the granulations, but whether the result of this is a degenerative change in the cells themselves,

or a modified exudation of liquor sanguinis, is uncertain. We know that it can be artificially produced by the repeated application of a sharp irritant, such as blistering fluid, and that after a time it will disappear of itself. The actual cause of its spontaneous origin is to be sought perhaps in the properties of the chemical products of decomposition of the exudation that escapes from the vessels of the granulations. The rational treatment of the ulcer is to remove all sources of irritation by protecting it from friction, and counteracting the effects of unhealthy discharges by the use of detergent dressings. The fibrinous rind is quickly reformed, hence its simple removal will not suffice for a cure.

**2. Fungous disease.** -There are two forms of this, the one depending upon an interference with the return of blood from the part, where it may be considered much after the nature of a local œdema; the other upon excessive formative activity and defective organising power of the granulations, kept up by continued slight irritation. Very often both causes work together.

The granulations are large, pale, and gelatinous, and discharge a thin muco-purulent matter. They may be so exuberant as to rise for some distance above the level of the margin of the ulcer, and even to overlap it. They have but little tendency to recede of themselves, and this is notably the case when the veins of the part are dilated and varicosed, and the margin of the ulcer is indurated. Besides the excess of watery exudation, there is softening of the granulations from mucoid degeneration: this is shown by the nature of the discharge. The treatment consists in removing all obstacles to the return of blood from the ulcer, by attention to position, by artificial support of the veins, and by applying pressure directly to the granulations: this relieves the fulness of the capillaries, and by



making the extravascular greater than the intravascular tension, tends to reverse the osmotic current, or, in more familiar but less explicit terms, favours absorption. The cells of the granulations, robbed too of a part of their blood supply, undergo fatty degeneration. Healing may be hastened by destroying the superficial granulations by an escharotic. Stimulant applications, as we have seen, promote a more fibrinous or plastic exudation, and this gives better support to the vessels. Astringents act both as irritants and desiccants; they diminish the calibre of the granulation capillaries more by extracting water than by increasing the functional contractility of the walls of the vessels.

**3. Hemorrhagic condition of the granulations.**—This may be solely due to the tension on the venous side of the capillaries being so great as to burst the delicate walls of the latter, as when the veins of the leg are dilated and varicosed, or the veinlets returning from the ulcer are compressed by its callous, indurated walls. On the other hand, the capillaries may be unable to withstand the normal pressure, owing to a primitive weakness in construction or fatty degeneration of their walls.

Then there are some ulcers that are essentially hemorrhagic, *e.g.* those due to malignant disease, scurvy, etc. The result of hemorrhage is to delay healing, especially when it occurs in the depths of the granulations, as well as on the surface, for vessels are not only destroyed by rupture, but some are obliterated by the compression of the extravasated blood.

In extensive burns, the granulations readily bleed; even the removal of external support involved in changing the dressings, or the mere dependent position of the part, may suffice.

**4. Diphtheria of the granulations.** This phrase is unfortunate in its application to the condition of an ulcer, for it implies that the latter is necessarily

the seat of true diphtheria, either by direct inoculation with the virus of that specific fever, or by infection of the wound through the blood. No doubt an ulcer offers a favourable surface for absorption, and especially when it is spreading; and there is no reason why there should be immunity from the action of the poison of diphtheria, nor why we should decline to recognise a true diphtheritic disease of the granulations. But what we wish to insist upon is, that the expression "diphtheria of wounds," as commonly employed, is used to describe in general terms the local appearances that closely resemble those following true diphtheritic infection, without the expressed or implied belief that in all cases such infection has really happened. It would certainly be better to restrict the application to the specific disease, diphtheria, but long usage stands in the way of this. The difficulty in arriving at a satisfactory nomenclature lies in the fact that many organisms possess the property of causing similar local manifestations, although each one differs from the rest in its real nature. Some pathologists consider the disease in question as identical with *hospital gangrene*. Billroth, whilst admitting a resemblance between the two affections, says they are quite distinct from one another, and expresses the belief that each one is dependent upon a specific poison. This coincides with our own views. Diphtheria, when it attacks an ulcer, gives rise to excessive fibrinous exudation, that infiltrates the granulations and forms a thick rind on the surface. The vessels of the granulations become thrombosed, and this aids in the molecular disintegration. The tissues around show inflammatory hyperæmia, and are in their turn destroyed.

*Other conditions of an ulcer :*

1. **Infamed ulcer.**—The usual signs of inflammation are seen in the skin around, and in addition there is often some catarrhal exudation, and



occasionally a number of small acute eczematous ulcers. All this will depend upon the severity and duration of the inflammation. If the ulcer has been previously healing, the process is arrested, and the marginal zone of newly formed epithelium is destroyed. The granulations lose their florid hue and change to ashen grey. The superficial layer at least is destroyed, for the circulation in the delicate capillary loops is quickly arrested. After this the surface of the ulcer is more or less smooth, and covered with pus and the *débris* of broken-down granulations. There is great pain, and tenderness. Should the inflammation continue, the ulcer spreads.

2. **Irritable ulcer.**—An inflamed ulcer is necessarily an irritable one, but the burning pain may be out of proportion to any visible cause. It is in some way due to the state of the nerves supplying the part, and is not always co-extensive with the ulcerated surface, but may be limited to a particular part corresponding to the known distribution of a nerve. By some it is supposed that the nerve fibres are exposed in the floor of the ulcer. Billroth suggests that they may undergo bulbous enlargement, like the nerve trunks involved in the cicatrix of an amputation stump; but this is mere surmise. It seems to us that the nerve-fibres are subject to irritation in one of two ways: either chemically by the discharge, or mechanically by being stretched in the base or margin of the ulcer. The latter is more probable, since it accords with the fact that the pain is sometimes limited to a certain area. There is usually evidence of slight inflammation. The granulations are very small, and the secretion is scanty. Whilst this condition lasts repair is at a standstill. It is noteworthy that circumcision of the ulcer, or subcutaneous division of the affected nerve, may at once remove the pain, and with it the cause that prevents healing (Hilton).

**3. Callous ulcer.** This is as much the consequence as the cause of delay in healing. It is mostly seen in varicose ulcers of the leg. The return of blood is prevented by the over full veins, and the result is that the granulations are deprived of their proper supply of arterial blood, and the tension in them is increased. Moreover, as these ulcers are situated in the front of the leg, they are more liable to friction; and as but little areolar tissue intervenes between the base and margin of the ulcer, and the underlying bone, adhesion takes place, and this prevents cicatrisation. The margin of the ulcer is thick and indurated, almost cartilaginous in consistence. It may be more than an eighth of an inch deep. It usually forms a steep declivity; it is rarely undermined. Microscopical sections of the walls show a coarsely fibrillated or homogeneous substance, in which leucocytes are embedded. The number of the latter varies inversely as the induration. The granulations at the base are unhealthy; they may be large and cedematous, or almost wanting. The papillæ of the surrounding skin are hypertrophied; they may be hidden by the exudation, or give a warty appearance to the surface. The skin itself is congested, and often deeply pigmented.

*Symptomatic ulcers.*

**1. Varicose ulcers.**—No other variety passes through so many phases of the ulcerative process. They are of great interest on account of their frequency, and the difficulty that attends their cure. We class them with the symptomatic ulcers, since their origin or extension depends upon a diseased state of the veins. They are almost confined to the lower extremity, for in this part varix of the cutaneous veins reaches its highest development; and, besides this, the leg is much exposed to injury, the skin is in close proximity to the bone, and therefore liable to

become adherent to it, and the force of gravity adds to the difficulty of the venous circulation.

*Mode of origin.*—Varicose ulcers begin in one of four ways:—

(1) By rupture of the attenuated walls of a dilated vein.

(2) By thrombosis of a cutaneous vein and its capillary tributaries.

Now there is but little vascular communication between contiguous capillary areas of the skin, and consequently the part, cut off from its direct supply, dies, and is cast off as a slough, or it undergoes molecular softening. In either case, a circular or oval ulcer is left.

(3) By an abrasion.

(4) By the gradual transition of eczema to ulceration.

The inflammation for a time causes only a catarrhal exudation, but sooner or later the true skin is exposed by the destruction of the rete.

If on the first appearance of a varicose ulcer it be kept clean and protected from injury, and the pressure in the veins be lessened by raising the limb, it will readily heal. On the other hand, the deleterious effect of decomposing discharge, the continual fretting, and the congested state of the ulcer and skin around, increase and perpetuate the impairment of nutrition. The venous distension is the chief cause of œdema of the granulations, of rupture of capillaries, and pigmentation of the skin. It also explains the failure of cicatrisation, for whilst it prevents healthy granulation, it gives time for the lymph effused into the base and edge of the ulcer to become indurated, and so to act as a secondary barrier to the circulation.

The inflammation that extends for some distance beyond the ulcer causes eczema of the skin.

Apart from the general treatment applicable to

any ulcer, the special indication is to support the vessels by pressure and position, and thus remove the prime cause of the ulcer spreading or remaining stationary.

2. **Tubercular (scrofulous, strumous) ulcers.**—Tubercle is connected with ulceration in three ways :

- (1) It may constitute the sole cause.
- (2) It may give rise to it through the medium of suppuration in subcutaneous structures—*e.g.* lymphatic glands and bones.
- (3) It may modify ulceration of the skin induced by other causes.

Sinuses, resulting from caries of bone, open on the surface by apertures, which appear closed by the granulations. The latter in some cases are fungous, and the mass everted ; but quite as often they are small and pale. In the latter case the ulceration sometimes spreads to a considerable distance, and has all the characters of a tubercular sore. The same may be said of the undermined ulcers of the neck and other parts where lymphatic glands have suppurated. Not uncommonly the inflammation first shows itself as an eczema ; but as a rule a small abscess forms in the subcutaneous tissue, opens and leaves an ulcer with undermined edges. This is very frequent over bony prominences, and other parts liable to friction.

Tubercular ulcers are very chronic. The base is smooth and dry, or bathed with an ichorous pus furnished by small unhealthy granulations. The edges are thin, and often widely undermined. The skin around is of a dull purple colour, from congestion. A probe may sometimes be passed for several inches beneath it. These ulcers occasionally spread over a wide surface, and when they heal leave unstable scars. When situated over bones and tendons (*e.g.* about the malleoli), their bases become

adherent to the parts beneath, and are much exposed to irritation from movement and friction. Healing may be indefinitely delayed in such cases, and when it does take place the surface is irregular from hypertrophied papillæ and the remains of over-grown granulations. This is known as a "warty cicatrix."

**Syphilitic ulcers.**—These may be divided into primary, secondary, and tertiary. The primary sores, or chancres, are of two kinds, infective and non-infective, indurated and soft.\* The latter terms are misleading in practice, for a soft sore may harden by local irritation or specific induration. A hard sore can be made to suppurate and spread by the irritation of decomposing discharges or artificial stimulation.

**Non-infective chancres** are often multiple, it may be from the first or from auto-inoculation. They form quickly. A pustule bursts, and leaves a small painful ulcer with sharply cut edge and purulent base. For a time it spreads. The virus contained in the secretion is highly irritative. Conveyed by the lymphatics to the nearest set of glands, it excites acute inflammation, which is likely to end in lymphatic or peri-lymphatic abscess. From general and local neglect these chancres occasionally spread extensively, and are then termed phagedænic.

**Infective chancre.**—A typical hard sore shows only an abrasion or fissure. The induration is very marked and circumscribed. A thin ichorous discharge exudes from the surface. It contains large clear cells with two or more nuclei, which some pathologists consider characteristic. We cannot confirm this view. The induration of the base of the ulcer is a sign of constitutional infection, and appears from seven days

\* It should be clearly understood that the terms "infective" and "non infective" are here used to indicate respectively the presence or absence of constitutional effects. Both hard and soft chancres are locally infective.

to seven weeks after inoculation (Lee). There is induration, and moderate enlargement of the nearest group of lymphatic glands, but not, as a rule, suppuration.

Between characteristic hard and soft sores there is every gradation in the local characters.

**Secondary syphilitic ulcers.**—These include ulcerated *mucous tubercles* and other superficial ulcers. Mucous tubercles denuded of epithelium are typical specimens of fungous ulcers. They are met with at the junction of skin with mucous membrane, especially about the vulva and anus. They are also found on the mucous membranes of the mouth, pharynx, and larynx : and on the skin, *e.g.* in the flexure of the groin, in those parts, in fact, where friction, moisture, and acrid discharge cause irritation. They are sessile masses of granulations which discharge a glairy muco-pus. They differ from piles (1) in encroaching more upon the skin, (2) in their being pinkish white, and not bluish in colour, (3) in being flatter. The mistake most likely to be made is that of confounding mucous tubercles, seated on external piles, with simple ulceration of the latter. Mucous tubercles, or *condylomata*, are distinguished from warts in that the former have overgrown papillæ fused together by swelling of the intervening tissues, whilst in warts the papillæ are free.

The **secondary ulcers of the throat and mouth** are met with chiefly on the tongue, soft palate, and tonsils. They appear at first as milky-white patches of epithelium soddened by exudation. On removal of the epithelium, superficial ulcers are left. These are oval or roundish in outline. They tend to get well of themselves, and are symmetrical.

**Tertiary syphilitic ulcers** commence by the breaking down of gummata, when they are deep and their edges undermined ; or by a more superficial lesion



of the skin and mucous membranes. They are less symmetrical than the early secondary ulcers, and are less likely to undergo complete spontaneous cure. They spread in a *serpiginous* manner. Whilst the part first affected is healing extension takes place at the margin, so that wide cicatrices are edged by bands of ulceration. This condition is chiefly found in the superficial variety.

Hutchinson says that "syphilis is the parent of all phagedæna." To this we cannot entirely subscribe, believing, as we do, that there are other causes that may produce it in wounds exposed to bad hygienic conditions. Phagedæna is a true ulceration. The tissues bounding the ulcer rapidly melt away as the ulcer spreads; hence it is chiefly to the margin that one's attention is directed.

A **sloughing ulcer** is a combination of ulceration and gangrene. Visible portions of tissue die, and remain attached for some time to the base of the ulcer, and their histological characters can still be recognised. (*Vide* Gangrene, pages 45-52.)

Primary syphilitic sores are occasionally phagedænic, both at the point of inoculation and as a sequel of lymphatic bubo. They destroy the tissues widely and deeply, including vessels such as the arteria dorsalis penis, and the large arteries and veins in the groin.

Tertiary phagedænic ulceration attacks the skin, throat, nose, and rectum. We have seen it spread over the greater part of the face and eat away the external ear. In the throat and rectum it may give rise to dangerous hæmorrhage.

**Typhoid ulcers** afford a good example of extension lesion, with but little cicatricial contraction. This is due to the rapidity with which the morbid process subsides. They contrast strongly with tubercular ulcers of the intestine, which are very chronic, and give rise to a puckering and narrowing of the gut.



Typhoid ulcers lie in the axis of the bowel, tubercular athwart it, the former affecting Peyer's patches and the solitary glands, the latter following the course of the blood-vessels.

Typhoid ulcers are sharply defined, and their margin is often shreddy and undermined. Tubercular ulcers usually have an indurated base and edge.

## CHAPTER IV.

## GANGRENE.

GANGRENE, or death of a visible portion of tissue, ensues whenever the circulation is permanently arrested, or so far impeded that the vitality cannot be maintained.

The dead portion is termed a *sphacelus*, or *slough*, and, in the case of bone, a *sequestrum*. It may depend upon one primary cause, as when a large artery or several small ones are blocked by fragments of clot detached from the interior of an aneurism. But more often several factors are combined in their action ; thus, in so-called spontaneous senile gangrene, the heart's action, perhaps, is weak from fatty degeneration, the large arteries roughened by atheromatous deposits, the medium or smaller-sized ones rigid and irregular from calcification of the middle coat, the capillaries obstructed by the pressure of some slight accidental inflammation, whilst the circulation through the veins may be impeded by varicosities, or by a difficulty in the blood traversing the pulmonary capillaries from bronchitis and emphysema.

**Dry and moist gangrene.**—The terms *dry* and *moist* as applied to gangrene are only relative, serving as types for the clinical grouping of cases which vary within considerably wide limits. The more the obstruction is on the arterial side of circulation, the drier will the necrosed tissue become ; for then, whilst the blood enters in a gradually diminishing stream, the continual evaporation desiccates the tissues until the skin is *mummified*, or hard, dry, and leathery ; on the other hand, if the arterial supply be free, but the transit through the veins and capillaries be

checked, the tissues are gorged with blood and soddened by exudation.

**Senile gangrene** sometimes starts from diffuse thrombosis of the tibial arteries and their branches. The low vitality of the walls of the vessels favours coagulation of the blood, and this is aided by the sluggishness of the current from (1) a weak propelling power, (2) the resistance due to increased friction against a rough surface, and (3) loss of elasticity. The coagulation, moreover, closes the circuits of collateral supply, and so the tissues are starved from want of sufficient nourishment. The force of the current is so much diminished that when the blood reaches the capillaries and veins it stagnates and thrombosis ensues. Such is the pathology of those cases of gangrene extending slowly over a wide tract of tissue.

In many cases the onset is due to some slight injury causing inflammatory thrombosis, which continues to spread far beyond the limits of the original seat of irritation.

The arrest of the gangrene is apt to occur near the joints, where the arterial supply is freer than elsewhere, and the anastomoses larger and more numerous.

**Changes in the dead tissues.**—Evaporation goes on after the circulation has stopped, and so a gradual desiccation reduces the part to a dry, hard mass. The red blood corpuscles break up, and thus set free the colouring matter, which rapidly diffuses, and becomes reduced in the general disintegration of the tissues, so that it appears in the form of black granules of hematin; hence the dark discoloration, which is increased by the iron of the blood combining with sulphur derived from the decomposition of albuminoid bodies.

The leucocytes undergo fatty degeneration, but

they persist much longer than the red corpuscles. The cells of the adipose tissue discharge their fat, which takes the place of the water lost by evaporation, acts as a perservative to the structures it infiltrates, and gives the translucent appearance on section.

The structural identity of the tissues is maintained for a long time, cartilage, tendons, ligaments, and muscles especially resisting the chemical changes that tend to obliterate their characteristic features. The medulla of bone perishes like other unstable matter, but the osseous framework remains intact. The fibres of striped muscle become dissociated, and split transversely into their sarcous elements; pigment granules are seen within the sarcolemma, derived probably from the colouring matter of the muscle. In nerves, the white substance of Schwann breaks up and liquefies; the primitive sheaths collapse around the axis cylinders, which are the last to go. The endothelial cells of the blood-vessels quickly disappear; next the muscular fibres and the adventitia; and finally the elastic coat. Crystals of stearic acid, cholesterine, etc., are formed by the decompositions of fat set free from the corpuscles or derived from metamorphosis of the tissues.

**How the necrosed part is got rid of.**—In the living tissue on the confines of the dead a layer of highly vascular granulations makes its appearance. It is seen on the surface as a red *line of demarcation*. The thrombi in the vessels and the products of decomposition of the dead structures act as irritants, and set up a limited inflammation which ends abruptly next the gangrenous part, but fades away into the healthy tissues.

The softening which begins as the consequence of serous and cellular infiltration is continued by the destructive action of the granulation tissue, and does not cease until the solution of continuity is complete,

or, in other words, until the dead part is set free from the living.

The bones and muscles are less liable to gangrene than the skin and subcutaneous cellular tissue, for they are fed more directly by the main arteries which lie near them. The threatened stoppage of the circulation is checked or averted by the high blood-pressure and free and large anastomoses. This is the explanation of the *conical*, or "*sugar-loaf*" stump left after the necrosed structures have been thrown off spontaneously.

**Moist gangrene.**—The *causes* of moist gangrene are (1) acute inflammation; (2) obstruction to the venous circulation, as when a large vein is wounded, or a bandage applied too tightly; (3) plugging of an artery which has few anastomoses, *e.g.* the middle cerebral. The result is a venous reflux, and a stagnation in the area of distribution of the artery; (4) localised occlusion of the main artery of a limb, as in some cases of ligature for the cure of aneurism. The blood reaches the distal portion of the limb by smaller vessels, which, being rigid from disease, cannot enlarge in time to set up a sufficiently free circulation, the obstruction is so great that there is not force enough to drive the blood through the veins and capillaries; (5) widespread primary capillary thrombosis, as in *cancrem oris*.

A good example of moist gangrene from inflammation is seen in phlegmonous erysipelas, in which the tension is so great that the capillary circulation stops from pressure without and thrombosis within, hence the sloughing of the skin and cellular tissue.

Free incisions provide a drain for the exudation, unload the engorged vessels, and remove the resistance of tense fasciæ.

Gangrene of the internal organs is necessarily moist, for evaporation cannot take place. If at the same time the air is excluded, as in large infarctions

of the spleen and brain, progressive molecular softening replaces ordinary putrefaction.

When the dead part is exposed on the surface of the body, or in the respiratory and alimentary tracts, there is decomposition with the evolution of stinking gases. In moist gangrene (of the leg, for example) the skin is cold, and purple from congestion and stagnation; not uniformly so, however, for there are mottlings from dilatation and rupture of small vessels, and streaks showing the course of the superficial veins. It pits on pressure, for there is a loss of elasticity, and a bogginess from serous infiltration.

Evaporation does not suffice to carry off all the fluid that exudes; hence the epidermis is raised in the form of bullæ which contain blood-stained serum, and later the gaseous products of putrefaction. Soon a soft emphysematous crackling can be felt, due to the interstitial liberation of gases too voluminous to be held in solution. The discoloration gets more diffused, for the blood corpuscles break up and discharge their pigment, which then infiltrates the tissues. Again, the vessels burst from softening of their walls and the pressure of inflation. The purple tint changes to green and black. The epidermis peels off in soft flakes. The skin and cellular tissue melt away, exposing tendons and fasciæ, and these, dissolving, leave the bones bare and discoloured. The sequence of events above described is usually broken by removal of the limb or by the patient's death. It is very difficult, often impossible, to tell where the gangrene will stop, or whether it will stop at all if left to itself, especially when it follows progressive thrombosis of the main artery and vein, or infective inflammation. Considering the danger of septic intoxication and infection, and the uncertainty of spontaneous arrest, it is better, in most cases, to operate early and wide of the disease.



**Hospital gangrene**, called also sloughing phagedæna, is a disease that attacks wounds. The circumstances known as "bad hygienic conditions," which favour the propagation of germs, conduce likewise to the development of this formidable disease. Nor does this view conflict with the opinion that individual predisposition of the patient has much to do with the etiology; and hence it can be understood why a man already broken down by privation, by long exposure to insanitary influences or exhausting disease (such as tertiary syphilis), should fall a prey to this malady sooner than one in the bloom of health.

The active cause, no doubt, is some virus introduced from without: but its ravages are more extensive and fatal when it meets with tissues of impaired vitality. We take it, then, that hospital gangrene is due to inoculation of a wound, old or recent, with an organism capable of causing rapid death of granulations, and in succession all the structures around and beneath them. The general system becomes infected, partly by the original poison, and partly by the products of disintegration of the tissues. Absorption from the wound goes on unchecked, for there is no barrier of granulations to prevent it. Although in some ways it resembles diphtheria of wounds, pyæmia, and septicæmia, it is probably distinct from them. The disease is sometimes sporadic, though not uncommonly it is epidemic. It is much rarer than it used to be, for the circumstances that conduce to its development have been to a great extent done away with. They are overcrowding of wards and camps with patients suffering from unhealthy wounds, want of sufficient means to cleanse such wounds, bad ventilation, and all other conditions that make up "hospitalism" in its most virulent form.

*Local changes.*—As the name sloughing phagedæna denotes, there is gangrene, with true ulceration or molecular disintegration. The disease is said to assume two forms, the “pulpy” and “ulcerative.” This probably depends upon the mode and rapidity of destruction of the tissues. When it is very rapid, and the vessels are extensively thrombosed, considerable portions die before they are cast off, and, whilst adherent, they form soft decomposing sloughs. If, on the other hand, the margin melts away as the disease extends, we have the ulcerative form. Between the two extremes there is every gradation.

The surface of the ulcer is either smeared over with pus and the *débris* of disintegrated tissue, or it is occupied by dirty grey flocculent sloughs. The margin is sometimes undermined; it is always steep. The skin in the immediate proximity has a dull purplish hue, for here the stagnation and thrombosis are extremely marked. Beyond this is the usual inflammatory hyperæmia, fading into the healthy structures. The outline varies; it may be of indefinite shape, but very often it is curvilinear, and either crescentic, circular, or trefoil-like. As far as local signs go, they are much the same as in phagedænic venereal sores.

**Noma, cancerum oris.**—Cancerum oris, or gangrenous stomatitis, is, in the majority of cases, the sequel of an exanthematous fever—scarlatina, measles, and typhoid. It is not certain whether it is the direct result of the specific poison in these cases, or due to the marasmic condition left after the virulence of the latter has been expended. It sometimes occurs in weakly children as a primary affection, and it may be induced by prolonged mercurialisation. It affects the gums and cheeks, and, very rarely, the jaws, as it continues to spread. I once removed a large portion of the maxilla that had necrosed.

In some instances it commences as a capillary thrombosis in the substance of the cheek, but more often as an ulceration of the mucous membrane, which extends widely and deeply in the surrounding tissues.

In a typical case, the skin in the centre of the gangrenous patch is converted into a coal-black eschar; next to this is a livid purple zone in which the blood has coagulated; then comes a ring of deep congestion, which fades away into the healthy tissue. On separation of the slough a perforation is found in the cheek, but, as a rule, the patient dies before this is effected.

In female children the genitals may be attacked, and this form is highly infective and rapidly destructive.

The ulcerative form is more often recovered from than that which begins as a primary parenchymatous gangrene.

## CHAPTER V.

## FEVER.

FEVER is marked by a rise in the general body temperature, and disordered function of the various organs. The average temperature of the body in health is 98.1° F., and this is pretty constant under widely different external conditions. If it be exceeded by one degree, the patient may be said to be feverish.

**Explanation of the pyrexia.**—Several theories have been advanced to account for this, but it may be safely said that each is too exclusive. We will take them in order :—

1. **Diminished perspiration.** — In simple fever the skin is hot and dry, which means that there is a check upon the cutaneous perspiration, or, in other words, a quantity of heat that should be extracted from the body by evaporation from its surface is retained. This of itself must of necessity tend to raise the temperature, but that it is not the sole cause is proved by the fact of a patient remaining feverish throughout prolonged perspiration. Thus, if the secretion of sweat be forced by pilocarpine during the hot stage of malarial fever, the temperature continues to rise for some time afterwards. This is not inconsistent with a fall of temperature during the sweating stage of this disease, and after the rigors of pyæmia. By a physical law, rapid evaporation must get rid of a quantity of heat, but the production meanwhile may be greater than the removal.

2. **Increased production of heat.** (A) Some have asserted that at the seat of local inflammation (say an acute abscess) the heat derived from increased

destruction of tissue raises the temperature of the part, and that the blood, as it courses through, is made hotter. This, too, is true as far as it goes, but then there is no constant ratio between the intensity of the local inflammation and the height of the general fever. A mere head of pus beneath the skin may increase the body heat by five or six degrees.

(B) The influence of the nervous system.--It is well known that there is a heat-regulating centre in the medulla oblongata, and that this may be disturbed in various ways: (*a*) through the cerebrum, as the result of emotional excitement; (*b*) reflexly, from irritation of the peripheral nerves; (*c*) from the action of pyrogenous matter absorbed from the seat of inflammation. The rise of temperature from cerebral excitement, though it may amount to several degrees, is very transient. Billroth disbelieves the reflex irritation theory. He says that if a wound be inflicted upon the foot of a dog, after all the nerves going to the limb have been divided, the temperature will still rise to the same degree as if the nerves had been left intact. This proves nothing; for the nerves cannot be divided without causing a wound, and the inflammatory products from this must be in contact with the central end of the nerve; besides, the operation itself must cause considerable irritation of the nerves. Other observers have shown that a rise in temperature is directly connected with nerve irritation. At a focus of inflammation the irritation may be mechanical, from stretching of the nerves, or chemical, from the action of the inflammatory products. In either case the stimulus is conveyed to the centre in the medulla, and there transmuted into a fresh stimulus, which is sent to the tissues throughout the body, exciting them to increased combustion.

(c) The action of toxic matter.--By toxic matter

we do not mean alone the products of putrescent decomposition of the tissues and discharges of a wound, but also the outcome of the chemical changes that take place in all cases of inflammation. The latter may be modified by decomposition, or the presence of a specific poison, circumstances which explain the great variety of fevers. Absorption goes on through the capillaries and lymphatics, and thus the blood becomes charged with poisonous material, which then acts upon the medulla, and probably on the other tissues through which it circulates.

**Other signs of fever.**—All the tissues of the body suffer more or less. This is shown by disordered function, and markedly in the *secretory organs*. The work done by them is much diminished. The blood is charged with noxious matter in a threefold way :—there is the morbid material furnished by the local inflammation ; to this is added the results of a general increase in the oxidation of the tissues, and the negative effect of partial arrest of secretion and excretion. Though many of the symptoms are in part due to the direct action of the pyrogenous matter on the organs which fail to carry out their functions, much is explained by the disturbed state of the vaso-motor, secretory, and trophic nerves of the *glands*. The first of these explains, to a great extent, the diminution in the watery constituents ; for, the lower the pressure, the less the filtration ; the second, perversion of the special function of the secreting cells ; and the third, disorderly metabolism of the tissue elements.

The *urine* is high-coloured, and of high sp. gr. ; it deposits urates. The water is scanty ; urea and uric acid are increased. The chlorides are diminished ; in acute pneumonia they are often absent. In simple fever the *perspiration* is diminished ; it is very acid, notably so in rheumatic fever. In this disease, and in hectic fever and pyæmia, profuse sweating is a



prominent symptom. The secretion of *saliva* is checked, and the discharge from the mucous glands is more tenacious than natural; hence the clamminess and thirst. There is more or less anorexia, perhaps vomiting. The furred condition of the tongue is an index to the state of the stomach. Constipation is the rule; but in certain fevers that have a specific effect on the intestines (*e.g.* cholera, typhoid, and some cases of septicæmia) there is diarrhœa. Whether there be flux or drought depends upon the vascular tension, the condition of the walls of the vessels, and the chemical and physical nature of the fluids that dialyse.

*The nervous system.*—There are symptoms directly referable to the nervous system, such as *headache* and *delirium*. These are explained rather by the pyrexia and toxic influence upon the brain than by the extent of vascular congestion; and hence the futility of excessive depletion by blood letting. Such treatment might do actual harm by adding the effects of anæmia to those of the fever. Sir W. Jenner has shown that in the specific fevers, and particularly in typhoid, headache ceases with the onset of delirium, whereas in tubercular meningitis it persists. The explanation is this: the poison of the specific fever at first excites the nervous system, and one of the chief signs of this is headache; but, later, it deadens the perceptive centres, and gives full play to disorderly and uncontrollable discharge of nerve energy—delirium. In tubercular meningitis the cause of the headache is twofold; there is the pyrexia, and the irritation of the brain by local inflammation. The latter is so intense that it is only when the exhaustion from the fever and subsequent compression has supervened that the headache is abolished. The physical conditions of headache are constant throughout the course of the two diseases; but by the time the

toxæmia of the specific fever has caused delirium, it has masked the headache.

The *muscular system*.—There is increased activity at the expense of diminished power. In slight cases this may not be evident during rest, but it becomes manifest on exertion; there is the sense and sign of weakness in the tremulous state of the patient. This is mainly nervous, but the state of the muscular fibres themselves (for pyrexia causes fatty degeneration) has some share in the process. When the fever is high or the toxæmia intense, and, above all, when the blood is suddenly charged with infective matter, the natural tonic contraction of the muscles is replaced by a succession of rapidly repeated contractions, over which the patient has little or no control. There are four degrees of this:

1. *Fibrillar tremor*.—This can be better felt than seen; like the subsultus tendinum, it indicates extreme nervous exhaustion.

2. *Rigor*.—Here the contraction is more pronounced and more general. It is accompanied by a sensation of cold which is subjective, for before the rigor sets in the temperature has generally risen several degrees. It is the relative coldness of the atmosphere to the heat of the patient's body. The mind remains clear, firstly because the disturbance is mainly confined to the motor centres, and secondly because the contractions are not sufficiently vigorous and sustained to fix the chest walls, and so cause cerebral congestion from asphyxia. There may be only an initial rigor; or a succession of rigors. They vary in degree from chattering of the teeth to shaking of the whole body. They are usually followed by profuse perspiration, not alone as a consequence of the rigors, but as a later link in the chain of toxic effects.

3. *Eclamptic convulsions*.—These in children take the place of rigors in the adult, for in them there

is greater excitability of the nerve centres. Not that adults are exempt from convulsions as the result of high fever. Primary convulsions usually usher in some acute fever, *e.g.* pneumonia; when they occur later some grave lesion should be suspected—cerebral embolism, thrombosis, etc. The contractions are more violent than in rigors. There is loss of consciousness and more or less asphyxia. There are many causes of convulsions, but here we are only considering those dependent upon the pyrexial state.

4. *Tetanic or tonic spasm.*—In reality it is clonic, but the contractions are so minute and follow one another so rapidly that without the aid of the myograph they appear fused. In a minor degree it is seen in “cramp” and “stiffness,” but it is the symptomatic essence of two pyrexial diseases, tetanus and hydrophobia. It probably depends upon a more continuous irritation than either a rigor or “clonic spasm.” The discharge of nerve energy is not so violent as in ordinary convulsions, and therefore the nerve cells are not so quickly exhausted.

## CHAPTER VI

## SURGICAL OR TRAUMATIC FEVER.

IN former times so constantly did general fever supervene on local injuries that it was believed to be a necessary consequence, hence the then universal acceptance of the expression "Traumatic Fever." We now know that patients may be subjected to the most severe operations, and yet remain entirely free from pyrexia. There are two conditions under which wounds are accompanied by fever; (*a*) tension of the part due to a large accumulation of blood, and inflammation, the result of the tension; (*b*) septicity, this is the efficient cause of nearly all cases of so-called traumatic fever, to be now described. For some little time subsequent to the infliction of the wound, the temperature of the body is generally lowered. This is due to the shock of the injury and the depressing effects of the anæsthetic. After an apyrexial interval, varying from twelve to thirty-six hours, the usual signs of fever show themselves; the heat of the body is increased; the patient looks somewhat flushed; he complains of feeling hot and thirsty; his appetite is indifferent, and his tongue moist and furred. In addition, there are headache and general restlessness.

The pyrexia varies within a range of three or four degrees; the temperature is rarely below 100 Fahr., or above 102·5°.

If we now examine the wound we shall find very much as follows: a certain amount of hyperæmia, incidental to the injury. The surfaces, which became glazed after the cessation of the bleeding, now look moist from effusion of liquor sanguinis from the dilated

vessels. There are, in fact, the signs of inflammation, and, other things being equal, the height of the fever is proportionate to the extent of the local disturbance.

**Cause of the fever.**—Allowing for the check on the cutaneous perspiration, and the possible effects of nerve irritation in the wound, there can be no doubt but that the general symptoms (the fever) depend mainly upon something absorbed from the wound: (1) Because whatever prevents a free escape of discharge causes an accession to the fever, and this in two ways; it affords a favourable condition for putrefaction, and increases the liability to absorption of the decomposed products. Per contra, the temperature falls on establishing efficient drainage. (2) Because the measures taken for keeping a wound aseptic minimise or altogether prevent the fever. The latter proves something more, viz. that the so-called traumatic fever is to a great extent a state of septic intoxication. In all wounds the injury destroys the vitality of a certain amount of tissue, and the necrosed structures are resolved into simpler compounds, but the continued presence of large sloughs in an aseptic wound is quite compatible with a normal temperature. In open and exposed wounds the dead tissue and the discharges undergo putrefactive decomposition, and furnish secondary products, which on being absorbed induce fever.

The **condition of the wound** favours absorption, for there are numbers of blood-vessels and lymphatics which take up fluids from the surface and transmit them to the blood and lymph streams beyond. As these vessels become firmly occluded by consolidation of the clots within and compression of the inflammatory exudation without, the fever declines, even whilst the wound is bathed with "discharges." A well formed layer of granulations is a strong protection against absorption, for the direction of the osmotic

current is away from the blood vessels, and granulations have no lymphatics.

Traumatic fever is generally more severe when it follows operations upon tissues indurated by chronic inflammation, for the vessels, being embedded in dense exudation, cannot collapse. To return to the course of the fever. In ordinary circumstances it subsides in a few days, it rarely lasts beyond a week. The temperature rises somewhat sharply at the beginning; for a day or two it oscillates about the maximum, with slight morning remissions. It terminates by lysis, or more rarely by crisis.

In some cases it is so slight that its existence is only revealed by the thermometer. When a large wound unites by the first intention fever may be entirely absent; hence, as Billroth observes, it may be considered as a "pathological accident." We would once more repeat that it is the result of local inflammation and absorption of decomposing secretions and tissues.



## CHAPTER VII.

## SEPTICÆMIA AND PYÆMIA.

ON attempting to define what is meant by the terms septicæmia and pyæmia one is met with this difficulty, that pathologists are by no means agreed as to the distinctive characters and relationships of the two conditions.

Some authorities make the word pyæmia cover all the cases of septic absorption from wounds or inflammatory foci that furnish products of decomposition, whether such absorption results in a general blood-poisoning with the occurrence of secondary metastatic infarctions and abscesses, or without them. They say that the variations observed in the symptoms, and post-mortem signs, depend upon the virulence or intensity of the poison, the state of nutrition of the tissues, and certain accidental circumstances likely to modify the course of the disease, rather than upon any specific character of the poison in different instances. Thus the absence of secondary metastatic lesions is explained by supposing that death or recovery occurs before sufficient time has elapsed for their development. We cannot admit this explanation, because, on the one hand, multiple secondary abscesses sometimes form very quickly after the injury or inflammation, *e.g.* the lungs may be full of them within a week of the onset of "acute necrosis"; and, on the other, uncomplicated fatal cases of septic poisoning are occasionally of much longer duration.

Dr. Burdon Sanderson looks upon septicæmia as a *non infective* process, or one in which there is no multiplication of the poison in the system. Since the poison necessarily becomes greatly diluted in the blood

and tissues, successive inoculations or injections from animal to animal produce less and less effect, or they may fail altogether. In any individual case the result depends (1) upon the power of resistance to the action of the septic material; some animals are more susceptible than others, and, *ceteris paribus*, the smaller the animal the smaller will be the dose necessary to kill it; (2) upon the strength of the dose; and (3) whether it be repeated or not.

The tendency is to recover, but the animal may be so overpowered as to succumb before the poison can be got rid of. The *materies morbi* consists of the chemical products of decomposition of organic matter. It is obviously such as would be furnished by a large wound (*e.g.* the placental surface of the uterus), and probably some of the cases of puerperal fever are of this nature. But if we thus limit the application of the term septicæmia, a large number of cases will be excluded, in which there is general blood-poisoning, also without secondary metastatic abscesses. It is customary to designate these by the expression "*septic infection*," in contradistinction to "*septic intoxication*" applied to the former group.

In septic infection all the signs and symptoms of septic intoxication, or simple septic poisoning, may be present; but there is this very important difference, that in the former the poison is multiplied in the system to an indefinite extent, and the disease can be communicated from animal to animal without any diminution in its virulence, in fact its intensity is often increased thereby. This is the nature of many cases met with in practice, and notably in post-parturient women.

The case with which puerperal fever can be induced by infinitesimal quantities of the infecting material is too well known.

There are three distinct forms of blood-poisoning : (1) *Septic intoxication*—sæpræmia (without metastases), from the absorption of the chemical products of decomposition of tissues and fluids, products incapable of undergoing multiplication in the system, and therefore of being transmitted from animal to animal with unimpaired virulence ; (2) *septic infection* (without metastases), in which the poison is not only multiplied, but to a certain extent developed in intensity as it passes from one field of cultivation to another ; (3) *pyæmia* or *septic infection with metastases*.

Simple septic non-infective intoxication, or the septicæmia of Dr. Sanderson, explains most of the cases of severe blood poisoning that recover without the formation of secondary abscesses. But it does not follow that all cases are of this nature ; nor, judging from the analogy between septic infection and malignant pustule, which is an essentially infective disease, and yet fatal in only about one-third of the subjects attacked (Greenfield), does it seem improbable that a certain number of patients may survive the death of the micro-organisms of septic infection.

**The nature of the poison.**—The immediate cause of the blood-poisoning seems to be due to the unorganised products of decomposition, and not to the organisms that determine the decomposition ; for the virulence of a fluid known to be intensely septic is not lessened by destruction of the organisms (Auder).

Several observers have shown that an amorphous substance can be extracted from the fluid that causes septic intoxication. Bergmann believed its composition to be definite, and he named it "*sepsin*" accordingly. Billroth doubts its specific nature, and thinks that there may be several products of decomposition capable of causing the symptoms.

Later observations have conclusively proved that the septic poison consists of one or more *animal*

*alkaloids* or *leucomaines*. (The term *ptomaines* is used to designate the alkaloids derived from the decomposition of dead animal matter.)

What is the cause of septic decomposition? Is the agency of living microscopical bodies essential to the process? Certainly, for in all decomposing animal fluids they exist in abundance, and Dr. Sanderson has shown that the products of decomposition capable of inducing septic intoxication are only formed in their presence.

What, then, is the reason of simple septic intoxication occurring in one instance, and septic infection in another? The organisms that set up ordinary putrefaction are destroyed by the living tissues, whilst those which cause septic infection have a much greater power of resistance. This of course implies that there is a different "germ" in each case.

Koch asserts that "organisms are not found in the blood of animals suffering from septic intoxication," but that they are invariably present in septic infection.

Is putrefaction necessary to blood-poisoning? Certainly not, if the evolution of stinking gases be taken as the indication and measure of it, as the following case shows: Mr. J. Lane amputated a thigh for disease of the knee joint with all antiseptic precautions. The discharges remained sweet, and the flaps to all appearance healthy, and *post mortem* no offensive smell could be detected in or about the wound. The symptoms were those of deep septic intoxication. There were no metastases.

Nor need the local irritation caused by the poison manifest itself in destructive inflammation, although the infection may cause secondary circumscribed and diffuse abscesses. The writer once lost a case of lithotomy, in which *post mortem* the wound in the bladder, which had nearly healed, appeared quite healthy. There were no signs of cystitis, peritonitis,

or pelvic cellulitis, or thrombosis of the local veins. The lungs were riddled with small abscesses, and there was a tract of diffuse suppuration between the scapular muscles in the back.

Yet although putrefaction, in the ordinary acceptation of the term, is not a necessary condition, it is so exceedingly common that it is safe to infer that the circumstances likely to give rise to it are much the same as those which underlie the occurrence of blood-poisoning, whatever form the latter may take ; whether it be simple intoxication, or intoxication with infection, or infection with metastases (pyæmia), or one or other combination of these phenomena.

Excluding the theory of spontaneous generation, and admitting that the organisms found in the discharges from wounds and in closed inflammatory foci are the cause and not the consequence of the decompositions attended with the formation of septic matter, and also that they are introduced from without, we can understand why blood-poisoning should be so closely associated with the overcrowding of wards, and the neglect of measures calculated to keep a wound healthy.

**The part played by the tissues.**— This is very important, both as regards the wound and the body generally. The greater the bruising and laceration, the more likely is a patient to suffer from blood poisoning. There are several factors that work to this end : (1) A considerable portion of tissue being killed outright, it quickly undergoes putrefaction if germ-laden air be allowed access to it ; (2) a large number of blood-vessels and lymphatics are opened, and thus an extensive tract for absorption is provided ; (3) short of actual death, the vitality of the tissues about the wound is greatly impaired by the injury itself and the inflammation it sets up.

Dr. Sanderson has shown that bacteria “are



incapable of producing the poison of septicæmia" (septic intoxication) "in the healthy organism." From this statement we must not infer that they do not gain admission to the body by the wound or some of the natural passages, but that the tissue elements destroy them, or are at least able to resist their action. Virchow long since observed that the animal tissues tend to get rid of matters obnoxious to them. What is called susceptibility or predisposition of the tissues to be affected by specific poisons (*e.g.* those of the exanthematous fevers) is only another way of expressing inherent weakness, or impaired vitality in a certain direction.

In erysipelas, which is an infective disease, the liability of communication from one individual to another is greatly influenced by the state of health. If two patients with similar wounds, the one a healthy young man, the other old and broken down by chronic kidney disease, be placed beside a case of erysipelas, the chances of immunity from infection, and recovery if infected, are more in favour of the former (De Morgan).

Further, closed abscesses and, notably, empyemata often contain the most fœtid pus laden with bacteria, and yet the symptoms of septic intoxication and infection may be almost nil.

Considering the minute size of the organisms, it is difficult to suppose that the wall of granulation tissue bounding the abscess is so germ-proof as to completely close all the possible paths of transit.

The explanation then turns upon the innocuousness of these organisms, or the vital strength of the tissues being able to destroy them. Thus much for the absence of infection. As to intoxication, the products of decomposition, though offensive to the sense of smell, must be taken as being not very strongly



pyrogenous, or as being so slowly absorbed that the tissues and excretory organs are able to get rid of them as fast as they pass into the system.

When the tissues have been undermined by long-standing disease, and especially when they enclose large collections of pus (as in a psoas abscess), they usually offer but little resistance to the absorption of septic matter. A patient may be apyrexial so long as such an abscess remains closed, but once opened there is an almost certainty of fever, perhaps of fatal intensity, unless the ingress of germs be prevented.

**Diagnosis of septic intoxication and infection.**—As septic intoxication is a concomitant of septic infection, the diagnosis of the latter must be made (1) from a knowledge of the transmission of the blood-poisoned state from one subject to another by means of a very small dose of the poison, or such as would appear inadequate to cause the symptoms, except on the theory of multiplication in the system (take *e.g.* a case of post-mortem inoculation with the fluid from acute peritonitis); (2) by the discovery of micrococci in the blood and secretions.

On the other hand, one would suspect septic intoxication alone where a patient becomes rapidly poisoned by absorption from a large surface of gangrenous tissue, and particularly if the escape of the decomposing fluids be prevented.

Pyæmia, or purulent infection, is accompanied by more or less septic intoxication, but the course of the fever is generally indicated by certain well-defined signs and symptoms.

On pathological grounds, septic intoxication and septic infection may be considered as distinct—the former being due to the absorption of poison generated locally by bacterial decomposition; the latter to the multiplication, local and general, of micrococci—and on clinical, so far as the power of transmission from one

patient to another is concerned ; yet, as the two conditions usually arise in similar circumstances, they may be described together as

**Septicæmia**, or “an acute blood-poisoning by the products of decomposition of the animal tissues and fluids; with or without the development and multiplication of infective organisms in various parts of the body.”

The exact mode of action of the poison is not well understood. In the main, it is probably chemical. The blood is profoundly affected, so that it is unable to nourish the tissues. At the same time, the tissues themselves are so altered as to be unfitted to carry on their healthy functions.

**Character and course of the symptoms.**—

The symptoms are chiefly referred to the nervous system, but the effects of the poison are manifested to a greater or less degree in the respiratory and circulatory organs and the alimentary canal. Cases differ somewhat from one another in the relative frequency and extent with which the different structures are affected. In the artificially-induced septicæmia of dogs the intestinal tract suffers considerably. The nervous symptoms are those of rapidly-increasing prostration and narcotism. The patient becomes apathetic and somnolent. In many instances there is low muttering delirium, gradually passing into coma. More rarely the mind is quite clear until the fatal end. Headache is not prominent. There are either no rigors, or only an initial chill that ushers in the other symptoms. Muscular weakness quickly supervenes. This is shown by failing power of the heart, the inability to sustain exertion, and, it may be, general tremor of the body.

The *temperature* varies considerably. Sometimes it is very high, 104° F. or more ; at others there is but little change ; or, again, it may be subnormal from

the first, and this often in the worst cases. Hence, taken alone, it helps very little in the prognosis. We do not find the steep fever curves as in pyæmia.

It may rise considerably after death. I have known it to reach  $108^{\circ}$  F. This is due to rapid destruction of the tissues, and the non-conversion of heat into functional power.

The *respirations* become quick and shallow from muscular weakness, and hurried still more, perhaps, from the pulmonary congestion and stasis.

The bowels are constipated in some cases, relaxed in others. Blood and mucus may be passed in the stools. There is sometimes uncontrollable vomiting.

*State of the blood and urine.*—If a drop of blood be placed on a slide, the red corpuscles will be seen to gather into clumps rather than rouleaux, but they show nothing definite in outline.

The white corpuscles are relatively and absolutely increased. Micrococci may be present.

The urine is high-coloured ; it is acid, but quickly becomes alkaline when passed. The urea and uric acid are increased. The phosphates, potash salts, and chlorides are diminished. Biliary constituents are occasionally present.

*Colour of the skin.*—The skin has a dusky earthy tint, or it is distinctly yellow. This may be from bile staining, but it is probably tinted with blood pigment set free by disintegration of the red corpuscles.

**Post-mortem signs.**—Rigor mortis sets in early and soon disappears. Decomposition is very rapid.

The *blood* coagulates imperfectly. It may be almost fluid. It is said sometimes to be tarry, but I have never seen this.

Disintegration of the red corpuscles goes on during life ; this is shown by the staining of the

endocardium and the intima of the vessels, observed directly after death.

The serum, too, is more or less deeply tinged, and dark from deoxidation of the hæmoglobin. Micrococci are sometimes present (septic infection).

The internal organs show marked congestion, especially at the most dependent parts.

Thrombosis is very common, and capillary extravasation far from rare. The latter is most marked in the mucous and submucous coats of the intestine, and beneath the serous membranes in the form of petechiæ or more diffuse extravasations. Meningeal hæmorrhage is less frequent.

There are several factors at work in causing the coagulation in the vessels: 1. A ferment is supposed to be liberated by the breaking up of the white corpuscles (Köhler). 2. The chemical composition of the blood is greatly altered. 3. Stasis from weak propelling power of the heart. 4. Swelling and shedding of the endothelium of the vessels, the débris obstructing the lumen. 5. Encroachment of the thickened intima upon the vascular channels. The hyperplasia is found in the small arteries ("obliterative endarteritis"), not in the venules; these, however, may show inflammatory changes secondary to thrombosis. 6. Aggregation of micrococci.

The mechanism of the hæmorrhage is thus explained: 1. The walls of the vessels are degenerated through (*a*) starvation, from the blood supply being cut off by coagulation; (*b*) fatty metamorphosis, from high temperature and the blood-poisoning. 2. Capillary thrombosis leading to venous reflux, and consequent rise of tension in the congested areas.

But "frequently the internal organs present no morbid appearances" (Billroth), or at least none that are characteristic. More often the above-described congestions, thromboses, and hæmorrhages, singly or

in combination, are met with, and as a rule the parenchyma is softer than natural from acute granulo-fatty change.

The *spleen* is enlarged, soft, and congested. The *liver* is greasy, of a dirty yellowish-grey colour. Its blood-vessels are full, especially those of the hepatic venous system. The tissue may be bile-stained. Crystals of tyrosin have been found.

The *kidney* is enlarged; the epithelium is granular and swollen. Exudation is occasionally seen between the Malpighian glomeruli and capsules. The sub-capsular stellate veins are injected, and the whole organ is more or less congested. There may be capillary extravasations between and into the tubes. The latter are often choked with epithelial debris.

The *lungs* are congested and oedematous. Patches of collapse and diffuse thrombosis, with or without extravasation, may be noticed.

The *pleura* and *pericardium* sometimes contain blood-stained serum. The *brain* is congested; more rarely it is the seat of meningeal extravasation. If the *skin* presented a dusky jaundiced tint during life, this will be found in the cadaver.

In septic infection groups of micrococci crowd the different tissues. They occupy the vessels and the intervascular spaces in the various organs.

The above-described symptoms and post-mortem signs may be taken as those of a constructive case of septicæmia, rather than as constant and necessary phenomena. Many of them are rarely absent, and most or all may be present. They are also found with undiminished intensity in acute pyæmia; but anatomically pyæmia is septic infection plus metastases.

**Pyæmia synonyms.** Purulent diathesis, purulent infection. Pus disease. Ichoræmia (Virchow). Pyohæmia simplex et multiplex, *i.e.* pyæmia with or



without metastases (Hueter). The last-mentioned designation corresponds to what we regard as septic infection and pyæmia respectively.

**History and current views of pyæmia.**—

The name pyæmia was given to the disease by Piorry. The supposed necessary dependence upon absorption of pus *sermed* indicated (1) by the constant association of pus formation and the characteristic lesions; (2) by the discovery of cells and granular bodies in the blood, which were believed to be derived from segmentation of connective-tissue corpuscles (the leucocyte migration theory was not then recognised).

We now know that, whether the pus cells do or do not pass back into the vessels, they come from them in the first instance. Probably the corpuscular and granular masses thought to be simply altered pus were in many instances decolorised thrombi, set free by disintegration. (*Vide* Thrombosis, pages 384–393.) Koch, who states that “pyæmia does not arise, as was formerly supposed, from the entrance of pus into the vessels,” looks upon the disease as a general one, accompanied by metastatic inflammations. This is the light in which we regard it, viz. that pyæmia is septic infection with metastases.

**Causes of metastases.**—Metastases may originate in one of three ways. (1) By the arrest of emboli charged with micrococci and the chemical products of their fermentative action; (2) by the micrococci contained in the circulating blood acting on tissues whose vitality has been lowered by injury or disease; (3) by the alkaloidal poisons generated by infective organisms in the wound and secondary abscesses. It has been already said that thrombosis constantly occurs in the veins at the seat of primary infection. The resulting coagula, which may be regarded as dead tissue, afford favourable conditions for the growth and multiplication of micrococci.



Softening of the thrombi ensues, and the disintegrated clots are swept into the circulation, and lodged in vessels too small to allow their further passage. Thus are easily accounted for the multiplicity, shape, and situation of the secondary abscesses. This is by far the most common origin of pyæmic metastases. But there are cases in which the secondary softening and suppuration covers extensive and irregular tracts of tissue, and in which no emboli can be found. From this it is reasonable to infer that the micrococci carried by the blood current settle down in combined action upon the more vulnerable parts, and in them give rise to thrombosis, disintegration, and suppuration, as in the primary wound. The secondary diffuse suppurative synovitis of joints may be accounted for in this way. Subcutaneous injection of sterilised tubercular matter excites inflammation in tissues affected with old tubercular mischief, and, judging from analogy, it cannot be denied that the chemical products of the fermentation due to the micrococci of pyæmia may suffice to account for the secondary lesions of the disease. The state of clinical and pathological knowledge does not enable us to assert that the poison evolved by the bacteria which induce putrefactive decomposition is capable of doing so.

#### **Course and character of the symptoms.—**

Pyæmia is a continuous fever with very decided intermissions. This seems to show that the pyrogenous matter thrown into the blood is largely derived from the foci of the secondary or metastatic inflammations, for the intervals and accessions appear too marked to be explained by absorption from the primary wound, or phlegmon, which may to all appearance be taking a steady continuous course. Billroth, however, believes that "extensive progressive inflammation about the wound must be regarded as the chief source of such repeated infection."

The fever is ushered in by symptoms of general malaise, or by a chill. The temperature rises before the occurrence of the rigor. Both are due to the same cause, viz. a charging of the blood with poisonous matter, which first induces increased metabolism of the tissues, and then disturbs the equilibrium of the motor centres. The sensation of cold is caused either by (1) the rapid elevation of the surface temperature, or by (2) "the blood being driven from the capillaries by the spasm of the cutaneous muscles" (Billroth). The heat of the body may rise to 105° F., or even higher; it is to some extent proportionate to the nervous irritability of the patient, and so is the severity of the rigor. At the end of this stage profuse perspiration occurs, and the temperature falls, sometimes below the normal, but even then it quickly rises again, so that the fever is practically continuous. In this way it differs from ague, which in some respects it greatly resembles.

The chills and exacerbations are repeated at varying intervals. This is characteristic of the disease.

The severity of the nervous and other symptoms depends in great measure upon the acuteness of the fever. In pyæmia from "acute necrosis" all the signs of septic intoxication are well marked. In the subacute and chronic forms the mind is generally quite clear.

Meanwhile the patient rapidly loses flesh, for whilst his tissues are being burnt up they are not replaced, owing to defective assimilation.

The countenance assumes a cachectic hue, the features are drawn, and are expressive of extreme exhaustion rather than acute suffering. Towards the end the tongue becomes dry and brown, and the tainted sweetness of the breath more marked. Bed-sores are common.

The *prognosis* turns upon (1) the possibility of

getting rid of the primary source of infection ; (2) the extent and situation of the metastases ; (3) the duration of the fever. Every day that passes adds to the probability of recovery.

**Changes in the wound.**—The suppurative inflammation at first increases, in direct continuity, and by the formation of abscesses at some little distance beyond. The pus is thin—ichorous, as it is termed. Later, the wound becomes drier. Any granulations that have formed are destroyed. The neighbouring veins are thrombosed, and on cutting into them they are found to contain clots in various stages of formation and disintegration. The nearest lymphatic glands are enlarged.

**Metastases.**—During life localised and diffuse suppurations may be met with ; especially in the joints, viscera, and the subcutaneous, subfascial, and intermuscular connective tissue. The rapidity with which these abscesses form is often remarkable. Then, again, there may be evidence of pneumonia, pleurisy, and pericarditis. In rarer cases the eyeball is destroyed, and cerebral abscess or suppuration in the arachnoid may ensue.

**Post-mortem appearances.**—Except in very acute cases, where during life the signs of septic intoxication were well marked, there may be nothing very striking to be seen post-mortem in the state of the blood ; and the internal organs do not show such extensive congestions, thromboses, hamorrhages, and softenings as described under septicæmia. But there are quite characteristic lesions in the form of multiple circumscribed metastatic abscesses, and red infarctions in various stages of softening and suppuration. These are for the most part wedge-shaped, and are then evidently the result of septic embolism. The contents of the abscesses consist of disintegrated tissue and blood clot, mingled with pus. They are most

frequently seen in the lungs, but they are not uncommon in the spleen, kidney, and other organs.

More rarely the abscesses are scattered, apparently irrespective of the course of the circulation. These, and the occasional diffuse suppurations, are probably the consequence of primary thromboses, and of the phlogogonous action of the poison upon tissues predisposed to inflammation. The same may be said of suppuration in the joints.

The primary wound will be found infiltrated with pus, and the veins filled with disintegrating clots.

Such is a typical case of pyæmia ; but the signs of septicæmia and pyæmia may be found in almost endless combinations.

**Idiopathic pyæmia.**—Sometimes numerous abscesses attended with other pyæmic symptoms are met with, without our being able to trace their origin ; but then the source of infection may be some poison that has gained access to the system by the respiratory tract, or through some minute breach of surface in the alimentary canal.

The *pyæmia of new-born children* is in almost all cases due to infection of the umbilical wound.

## CHAPTER VIII.

## MICRO-ORGANISMS AND SURGICAL DISEASES.

THE vegetable organisms which affect the human body may for clinical purposes be classed under three heads :—

(1) The true parasites which possess a purely local action. Of these the most important are (*a*) the fungi found on the surface of the body in certain cutaneous disorders, *e.g.* ringworm; and (*b*) the plant which causes the diseased condition termed actinomycosis, to be afterwards described (pages 89-91).

(2) The organisms which thrive in putrescible fluids and tissues and in them set up a fermentative change with the evolution of stinking gases, and the formation of chemical substances having local irritative and general toxic properties. They are known under the names bacteria, saprophytes, and “carrion fungi.”

(3) The organisms which invade the living tissues and develop in them specific infective processes. The following discussion will be confined to the two latter groups. Taking these together, it may be affirmed generally that they are devoid of chlorophyll, absorb oxygen, and excrete carbonic acid.

The term **bacteria** is sometimes used in a way to cover groups (2) and (3), and sometimes in a more restricted sense, to include only the organisms which induce putrefaction as indicated above. Cohn employs the former method as a basis of classification and divides the bacteria or schizomycetes according to their shape into (*a*) microbacteria (bacteria proper), (*b*) sphaerobacteria (micrococci), (*c*) desmobacteria or filobacteria (bacilli), and (*d*) spirobacteria (spirilla). This

plan will be adopted here, as being the simplest and most comprehensive (Fig. 2).

The **microbacteria** or bacteria proper are cylindrical rods, the length of which does not exceed double their breadth. They are mostly single or arranged in pairs, rarely forming chaplets or strings, thus contrasting strongly with bacilli. Very frequently they are massed together in a copious gelatinous substance (zoogloea). They are motile, the movement being of an oscillating or rotatory character. They

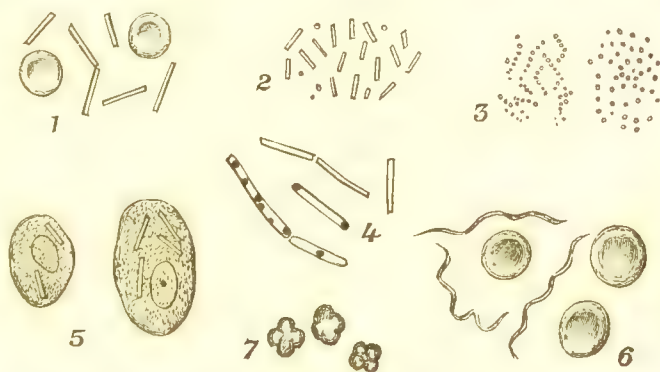


Fig. 2. —Various Forms of Bacteria ( $\times 500$ ). (After Ziegler.)

1. *Bacillus anthracis*, with red blood cells. 2. *Bacterium termo*. 3. *Micrococcus septicus*, separate and in chaplets. 4. *Bacillus malarie*, with spores. 5. *Bacillus leprae*. 6. *Spirochæta Obermeyerii* (spirillum of relapsing fever), with blood cells. 7. *Sarcina ventriculi*.

are easily destroyed by heat and chemical reagents, such as perchloride of mercury, iodine, etc. So far as is known their mode of reproduction is simply by cleavage or subdivision. Whilst they are the active agents in setting up putrefaction in dead matter, they soon perish when brought into contact with the circulating blood and living tissues, and in this way they differ notably from micrococci and bacilli.

The conditions requisite for their development and activity are the presence of dead animal matter,



oxygen, moisture, and a suitable temperature—that of the body being very favourable. The question whether bacteria are simple fungi which have developed virulence in putrescible tissues and fluids, or are specific organisms *ab initio*, is undecided.

A simple way of demonstrating them is to filter decomposing pus through wet blotting-paper on to a glass slide. The drop of fluid obtained should be evaporated to dryness by gentle heat. The dried material is then to be stained with logwood or methyl-violet for about half a minute. The staining fluid is now poured off and the slide again heated to dryness. Warm Canada balsam is to be dropped on to the coloured spot and a cover slip placed over it.

The **sphærobacteria** comprise two genera: micrococcus and sarcina. Micrococci are extremely minute spherical bodies, being not more than  $\frac{1}{25000}$  to  $\frac{1}{10000}$  in. in diameter. They may occur singly, but frequently they are grouped in pairs (diplococci), or in chaplets (streptococci), or in clumps or colonies (staphylococci). They do not possess spontaneous movement. They propagate by cell division. Unlike bacteria proper and bacilli, they do not elongate into filaments. The solid tissues and the lymphatic vessels are their chief habitats. This form of organism is found in septicæmia, erysipelas, infective osteomyelitis and periostitis, infective endocarditis, diphtheria, and other specific diseases.

The **desmobacteria**, filobacteria, or bacilli, are rod-like bodies. Whilst they resemble the microbacteria or bacteria proper in their individual shape and longitudinal growth, they differ from them (1) in that their length is at least double their breadth and frequently many times exceeding it; (2) in that, though sometimes motile, they are usually stationary; (3) and in that they multiply by the formation of spores as well as by cell division.

The chief diseases with which they are identified are malignant pustule or anthrax and tuberculosis.

The **spirobacteria**.—The only organism of this group that possesses any clinical significance is the spirochaeta (spirillum) Obermeyer's, which is found in the blood of patients suffering from relapsing fever. They have long, closely-set spirals. They are present only during the paroxysms of the fever. This would seem to show that the fungus propagates by spore-formation, and that certain stages of its development correspond to the successive intervals of the fever. Their movement in the blood stream is so active that occasionally they may be seen to displace the red corpuscles in their course.

As regards the property of spontaneous movement it may be said that the microbacteria or bacteria proper and the spirobacteria are motile; that the sphaerobacteria or micrococci are motionless; and that the desmobacteria or bacilli are usually so, although occasionally some forms are observed to oscillate.

**Biology of micro-organisms.**—There are constantly large numbers of microscopic organisms passing in and out of the body without causing any harm, and on this account they are termed non-pathogenic in contradistinction to others which are known to set up morbid action in the tissues and juices of the body, and are hence designated *pathogenic*.

To get some definite picture of the method in which pathogenic fungi act banefully on living animal tissues and fluids, it may be useful to sketch briefly some of the *conditions of life* of vegetable micro-organisms in general. In the first place there are certain requisites for their active development and functional activity. Amongst these are (1) suitable nutritive material in the form of inorganic salts and organic animal and vegetable matter to furnish carbon, hydrogen, and

especially nitrogen. (2) Moisture is another essential condition. It is a well-known fact that the presence in excess of the proper nutrient substances of an organism, by withdrawing water, will put a stop to its growth; thus sugar in large quantity prevents preserved fruits from undergoing fermentative change. Some organisms require more water than others; *e.g.* mould fungi can thrive where there is little moisture, whilst the yeast plant and bacteria only do so where it is abundant. (3) Free oxygen is necessary in almost all instances, and absolutely so in many. The bacillus anthracis cannot develop without it, although its filaments, and more notably its spores, may exist, so to speak, in a state of suspended animation for months or years even though completely desiccated. The development of malignant pustule and anthracæmia in tanners and wool-sorters is evidence of this. Those organisms which are dependent on oxygen for their active growth are called *aërobious*, whilst those that can do without it are termed *anaërobious*. According to Pasteur, bacterium termo, the active organism of putrefaction, if otherwise so placed as to be able to set up fermentation can develop in its absence. It is certain, however, that for putrefaction to proceed actively free oxygen must be present. (4) Although varying through a wide range of *temperature*, micro-organisms perish under the extremes of heat and cold. Fluid containing the bacterium termo becomes sterilised if kept at 65° C. for some time. In the case of those organisms which produce spores, the latter are less easily destroyed by heat than the rods or filaments. At a temperature of 5° C. all organisms cease to grow (Ziegler) although they may still survive.

**The presence of foreign or non-nutritive substances** is of great importance. Some do not act harmfully, whilst others, *e.g.* bichloride of

mercury, are positively germicidal. It is an interesting fact that in many cases the product of the fermentative process set up by an organism may prevent further development and activity of the fungus ; thus in vinous fermentation when the resulting alcohol is in quantity the yeast plant ceases to grow. Judging from analogy, it is reasonable to infer that the organisms of some of the specific diseases, *e.g.* scarlet fever, may generate a substance in the body which is inimical to their vitality. It is true the definite course of many specific diseases may be explained in another way, viz. that in a given time the organism may have exhausted the pabulum on which its continued vital activity depends.

If different organisms be present in the same nutrient fluid, some may act detrimentally to others, either by reason of their having a greater affinity for the nutrient material, or because their conditions of life are incompatible. If the agents of lactic acid, vinous, mould, acetous, and putrefactive fermentations, be all put into a solution of sugar, we can under certain conditions—several of which are naturally consecutive—obtain the corresponding fermentations in the order given, although they will not go on simultaneously. The germs exciting pathological phenomena are in some instances mutually opposite as regards their conditions of life. Thus whilst *bacterium termo* perishes when brought into contact with living tissues and fluids, the *bacillus* of anthrax is destroyed when placed in putrefactive media.

A modification of the nutrient material in quantity, or quality, or both, may explain why, if two individuals be exposed to the poison of a specific disorder at the same time, one may escape and the other not ; and in the event of both being infected, why the disease should develop with greater severity in the one case than in the other. Again, it is known that

the virulence of the specific fevers, *e.g.* scarlatina, varies in different epidemics, so that we have what are termed severe and mild types of the affection. This variation in the morbid action of the virus may be due to the pathogenic organism meeting with circumstances more or less favourable to its full development.

**The changes induced in the nutrient media** by the agency of microscopical organisms is of the highest importance from a pathological point of view. It is now generally conceded, both in the case of putrefaction, and of the chemical changes taking place in living tissues and fluids when attacked by pathogenic fungi, that the process is primarily and essentially one of vegetative fermentation. On this hypothesis the organisms in question are termed *formed or organised ferments* in contradistinction to the inorganised ferments, *e.g.* ptyaline, pancreatine, etc. As the ferments develop they abstract chiefly nitrogen, carbon, hydrogen, and oxygen, from the nutritive fluid, besides mineral constituents. The products of the fermentation are simple and definite in some instances, as when sugar is converted into alcohol. In others, especially when the nutritive material is richly nitrogenous, they are more varied and complex. Thus in putrefactive decomposition effected by the agency of bacterium termo, besides numerous nitrogen bases—leucin, tyrosin, amines, etc.—we get bodies termed *ptomaines* or *cadaveric alkaloids*. The poisons contained in tinned meats are believed to be of the nature of the latter.

The formed ferments can set free inorganised ferments; and probably the toxic symptoms in septicæmia are dependent not alone on the poisons primarily developed, but also on the products of the decompositions superinduced by the agency of these inorganised ferments.



The primary products of putrefactive fermentation of albuminoids readily undergo further changes if the supply of oxygen is plentiful. On the other hand, if it is deficient, we find sulphuretted hydrogen, indol, etc., in excess.

There is good ground for the belief that micrococci do not give rise to putrefaction.

The experimental researches of Pasteur and others have demonstrated the fact that some organised pathogenic fungi can, by special conditions of cultivation, be endowed with the property of causing other than their usual kinds of fermentation. This phenomenon is capable of explanation in one of two ways: (1) either the methods employed serve to bring into activity latent potential properties of the organisms; or (2) the organisms have not a fixed individuality. The theory of the "mutability of bacteria" (which assumes some primary forms or species ready, in favourable circumstances, to pass by transitional stages into other recognised varieties) is, in the author's opinion, based on errors of observation natural in the examination of such minute bodies. Weight is lent to this opinion by the fact that, in some infective diseases (*e.g.* anthrax, tuberculosis) the organisms, which are large, always possess the same physical features, and cause the same pathological changes after repeated cultivations in nutritive media.

By varying the conditions of cultivation a virus may acquire greater or less virulence. It may become more active, or be rendered so attenuated that it is capable only of slowly exhausting the media in which, in its more potent state, it would develop with vigour. The *protection* obtained by repeated inoculations with the attenuated poison—depleted or marasmic organisms—of hydrophobia and anthrax, is possibly thus explained.



**On immunity and susceptibility of the living tissues and fluids to the action of microscopical organisms.**—Microscopical fungi are widely distributed in the air, water, and in solids taken as food, and must constantly gain admission to the body in vast numbers. Why, then, do they develop within it with such comparative rarity? Many do not find a fitting soil (non-pathogenic bacteria), and consequently are either destroyed, deposited, or excreted. The conditions necessary for their development are: (1) the vital properties of the organisms themselves; (2) a predisposition on the part of the system of the recipient. Living healthy tissues are hostile to bacterial growth, but they may not be able to prevent it. Anything which depresses the vitality of the animal organism favours the inroads of pathogenic fungi, hence we find them selecting by preference tissues whose circulation happens to be defective—tracts of diminished resistance. In wounds and necrosed tissues—the maximum of diminished resistance we have the essential field for bacterial putrefactive fermentation. There are some variations of susceptibility which, although proved experimentally, are not as yet explained. These have reference to the species of animal inoculated, and age. As far as is known, the spirillum of relapsing fever will only develop in human beings and monkeys. Mice easily become infected with the virus of anthrax, while rats enjoy great immunity from it.

As regards the specific fevers, most of them select the periods of childhood and youth. Typhoid has a preference for young adults. Tuberculosis is mostly met with before mature manhood, or late in life, when the “second phthisical wave” sweeps over tissues in a state of senile degeneration. In some cases a previous attack confers greater or less immunity, in others

none. Protection when obtained may, as before remarked, be due to the exhaustion of the pabulum or nutritive matter necessary to the development of the organism. It is possible that the product of the fermentative action of the fungus may act as a deterrent against further fermentative action. The protection may be abiding, as in syphilis and small-pox ; or very transient, as in erysipelas.

**Changes effected by the organisms in the tissues and blood.** — Micro-organisms act deleteriously : (1) by withdrawing nutriment from the system ; (2) by abstracting oxygen, and thus interfering with the normal metabolism of the tissues ; (3) by developing a poison in their own nutrient material, which may cause toxic symptoms, as in septicæmia ; (4) by attacking the protoplasm of the tissue elements, and so altering their nutritive, formative, and functional activity ; (5) by setting up inflammation, either directly by their own irritant properties, or secondarily by means of unorganised ferments set free during and owing to their development and growth. It has been shown, that if several *non-pathogenic* ferments are present at the same time, one will develop to the detriment of the vital activity of the others, and, it may be, to their destruction ; and it is not unreasonable to suppose that a *pathogenic* ferment may in like manner act upon the living tissues, which may be likened to aggregations of organisms. The equilibrium of normal metabolism may be disturbed, in consequence of the pathogenic ferments causing different dynamic and chemical changes in the tissues and fluids of the body respectively. If this really obtains, there will of course ensue a corresponding disturbance of healthy function. Each fungus has a proclivity to attack certain groups of tissues, and the outcome of this selective affinity is the so-called *specific lesion* of the disease. Some pathogenic fungi (*e.g.* the bacillus

anthracis) flourish best in the circulating blood; whilst others (*e.g.* septicæmic micrococci, and the bacillus tuberculosis) choose in preference the solid tissues for their ravages. The micrococcus of erysipelas invades especially the lymphatics. The inflammation caused by the germs or their fermentative products may



Fig. 3.—Nodule containing Actinomyces from the Tongue of a Cow.

(From Ziegler after Sims Woodhead,  $\times 300$ .)

*a*, Central core: *b*, radiating club-shaped bodies: *c*, epithelioid cells in the granulomatous nodule: *d*, formative cells and new fibrous tissue.

result in acute suppuration and necrosis, as in infective osteomyelitis; or in the formation of granulation tissue, as in tuberculosis. It is said that the accumulation of inflammatory cells may bar the further growth of the fungus. This hypothesis would explain the obsolescence of tuberculous nodules.

## ACTINOMYCOSIS.

**Actinomyces** is a disease due to a vegetable parasite called *actinomyces*. Bollinger described the disease in cattle in 1877. About the same time Israel published two cases affecting the human subject, but he regarded the disorder as a kind of septicæmia. Ponfick was the first to recognise its identity with the parasitic disease of cattle (Fig. 3). It is a progressive inflammatory affection set up by the irritation of the fungus. The latter, which belongs

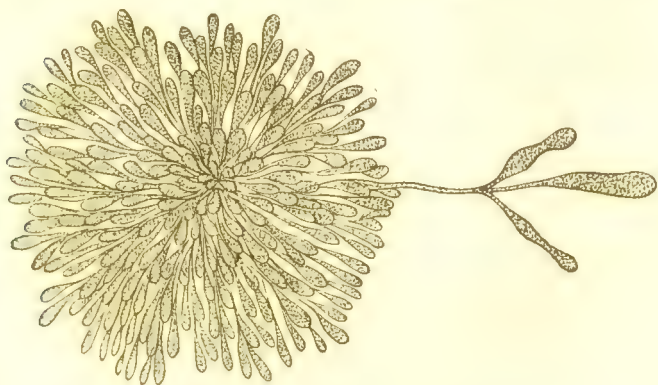


Fig. 4.—Perfect Form of Actinomyces. (After Ponfick.

(From *Reference Handbook of Medical Sciences*.)

A long filament terminating in bulbs is given off at one side.

probably to the hyphomycetes, is very characteristic. It grows in a ray-like manner. Microscopically, it is seen to consist of thread-like processes (*mycelium*) which give off bulbous swellings (*conidia*). Sometimes the bulbs are arranged in a moniliform series, or a thread of the mycelium may give off numerous bulbs (Fig. 4). The agglomeration of bulbs of this *ray-fungus* forms a mulberry-shaped mass of a yellowish colour, and about the size of a millet seed (Figs. 4 and 5). The bulbs are embedded in granulation tissue,

which in some parts breaks down and forms abscesses, whilst in others it organises into cicatricial tissue. Groups of the fungus granules, together with the surrounding inflammatory material, form nodular masses (Fig. 3), the size of a man's fist or even larger.

In the earlier stages of the disease the fungus exists as *cocci*, from which, later, fine mycelial threads are thrown out.

The recent observations of Kanthack show that

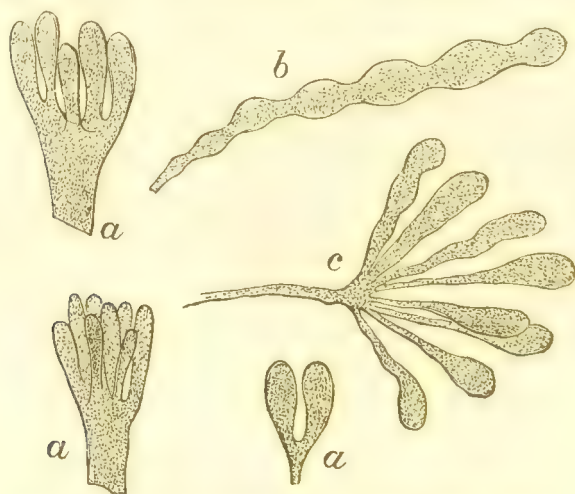


Fig. 5.—Various Forms of Actinomyces. (After Ponfick.)

(From *Reference Handbook of Medical Sciences*.)

*a*, Peculiar formation of bulbs; *b*, beaded form; *c*, branching of bulbs from hypha.

the *rays* are not invariably present. This authority regards them as degenerative products reacting like hyaline.\* The disease is most common in cattle and hogs. It is rare in man.

It usually starts in the alveolar process of the jaw, the fungus attacking a part where probably there is

\* *Lancet*, 1894, vol. i. p. 152.



solution of continuity of healthy tissue. From the jaw it spreads to the neighbouring parts. The tongue is often involved and enormously enlarged. The tissues of the neck, pharynx, œsophagus, and even the walls of the stomach may be invaded. "In man the soft parts of the neck, the lungs, and chest walls are often attacked."

The irritation of the bones causes side by side destructive caries and extensive osteophytic growths. Before the real nature of the disease was known in cattle it was described as "medullary sarcoma of the jaw," "osteosarcoma."

By the breaking down of the diseased masses, irregular, isolated, or inter-communicating cavities result. The ray-like bodies in the discharge from the abscesses were formerly looked upon as pathognomic, and assuming that Kanthack's view is correct, they may still be considered as singularly suggestive of the disease. They cannot, however, be relied on for diagnosis, since, as before said, they are sometimes absent.

#### MYCETOMA.

**Mycetoma (Madura foot, fungus disease of India).**—As regards its origin this disease is closely allied to actinomycosis. There are two varieties, the *white* or *orchroid*, and the *black* or *melanoid*. In the former, cavities are found about the size of a pea. These cavities contain granules resembling *fish-roe*. In the latter, hard, brittle, black bodies exist, varying in size from particles of dust to masses as large as a marble. It is a disputed point whether the black variety is merely a later stage of development of the white, or whether the two are distinct. In the white or orchroid form there are crescentic bodies, and in these are mycelial filaments and nail-shaped pigment granules. At the margin of



the crescentic or reniform bodies is a radial zone in which are club-shaped structures recalling to mind the bulbs in the rays of actinomycosis. Outside this radial zone is a layer of granulation cells begirt with cicatricial tissue.

The fungus causes marked hyperplasia, on the one hand, and caries of the bones and necrosis of the soft tissues, on the other. The foot becomes greatly enlarged and deformed. Abscesses open on the surface through mammillated crateriform apertures.

The disease is certainly due to the presence of a fungus, which consists of a fine mycelium producing bulbs. The "giant bulbs" of mycetoma are generally much larger than the corresponding bodies of actinomycosis, but in each disease the bulbs vary greatly in size in the same and different cases. The name *Chionyphe Carteri* has been given to the fungus of mycetoma.

## CHAPTER IX.

## ERYSIPELAS.

ERYSIPELAS is generally described as existing under three forms: (1) Cutaneous erysipelas; (2) phlegmonous or cellulo-cutaneous erysipelas; (3) cellulitis; though it is certain that the poison is not the same in each case.

Those who still believe in the unity of the disease explain the different results, (1) by the mode of introduction of the virus; (2) by the degree of its concentration; (3) by the extent to which the cutaneous and subcutaneous tissues are predisposed to its action; (4) by the fact of one variety taking on the characters of another. This is especially the case in the cutaneous and cellulo-cutaneous forms. The former may end in suppuration, and the latter spread superficially as a capillary lymphangitis.

The theory now generally accepted, however, is that cutaneous erysipelas and phlegmonous erysipelas are each due to a distinct organism; and that whilst the virus of cellulitis is not yet fully determined, there are reasons for suspecting that it is not constant, or, in other words, that different organisms may originate the morbid process.

The micrococci of phlegmonous erysipelas are larger than those of the cutaneous form. They are arranged in chaplets, and instead of being chiefly limited to the periphery of the diseased structures are distributed equally through all parts. Finally, cultivations of micrococci derived from one variety do not on inoculation give rise to the other.

**Simple cutaneous erysipelas** is generally due to inoculation, or infection of a wounded surface

(Billroth, Trousseau); for the so-called idiopathic variety usually attacks the exposed parts of the body, particularly the face, or those most subject to scratches or abrasions, which may be so small as to pass unnoticed.

The poison is not highly diffusible through the atmosphere, but there can be no doubt as to this mode of transmission, as shown by the occurrence of epidemics and sporadic cases.

That it is of the nature of an exanthem is proved by there being a period of incubation, followed by an efflorescence or rash. It is peculiar in taking an irregular course, and in not conferring protection from subsequent attacks, or only for a short period.

**Morbid anatomy.**—Erysipelas may be defined as a specific superficial capillary lymphangitis. It is essentially due to a micrococcus which can be cultivated artificially. Even after many cultivations the micrococci are capable of setting up the disease. The nearest lymphatic glands are always enlarged and tender, though they rarely suppurate. The swelling may be noticed in them before the rash is manifest.

The affected skin is of a bright red or pink colour. It is swollen and oedematous, so that pressure causes decided pitting. The margin of the eruption is generally well defined, and raised above the surface of the healthy skin, though at times it is indistinct and difficult to differentiate from that of simple erythema; but then the diagnosis can be made by the constitutional symptoms which are quite pronounced, whereas in erythema there is little or no general disturbance.

The part is tender to the touch.

The exudation which causes the skin to feel firm and inelastic may be confined to the interstices of the subcutaneous tissue and the papillary layer; or raise the epidermis into vesicles which, by their coalescence,

form bullæ. The fluid in the vesicles is usually clear and pale, occasionally it is blood stained; this depends upon the degree of vascular tension and disintegration of the red blood-corpuscles. The vesicles and bullæ dry up or burst, leaving scabs without ulceration.

As the inflammation spreads at the periphery, it dies away in the parts first involved. This would seem to prove that the poison had worn itself out, or that the tissues had become insusceptible to its further influence.

When the cellular tissue is very abundant and lax suppuration may occur; this is most common in the scalp and eyelids.

The surface of the wound (if one exists) looks unhealthy, greyish, and discharges but little pus. Desquamation ensues on the subsidence of the inflammation, and the skin is left slightly stained.

As before stated, erysipelas may attack the mucous membrane of the mouth, nose, and pharynx. It then causes considerable œdema, and there is danger from swelling about the glottis.

**Microscopy.**—The skin is infiltrated with leucocytes, and the subcutaneous cellular tissue fibres are swollen, and more or less dissociated by the exudation.

Where the morbid changes are active, the lymphatics are said to contain micrococci (Lukomsky).

The lymph spaces and vessels may be choked with wandering cells. The white corpuscles while contained within the vessels have been found enlarged, and highly granular and refractive.

Bouchard has shown that the vesicles of erysipelas are crowded with spherical organisms. According to Koch, the micrococci are not found in the blood-vessels.

The **general symptoms** appear, as a rule,

before the local—twenty-four hours, or less. At the commencement, the patient experiences a sensation of cold; more rarely there is an initial rigor, and in children convulsions. The temperature rises to 104° Fahr., or higher. There are no marked exacerbations, unless the disease takes an irregular course with relapses. In the old and very young, and in those exhausted by suffering, the symptoms may be asthenic from the first. The duration of the fever is indefinite.

**Post-mortem appearances.**—Locally, as above described. Internally nothing may be observed beyond the general signs of continued high fever.

In very malignant cases there may be evidence of acute blood-poisoning—septicæmia (*q.v.*).

The small vessels of the lung (Busk) and of the brain (Bastian) have been described as containing masses of granular leucocytes.

**Erysipelas associated with septicæmia.**—This is a coincidence, and not a transmutation of two distinct diseases.

Erysipelas, by seriously impairing the vitality of the tissues, renders them very susceptible to the action of poisons capable of setting up other infective processes. It is well seen where puerperal fever develops in women who have been exposed to the infection of erysipelas.

**Erysipelatous lymphangitis.**—We have said that cutaneous erysipelas is always and essentially a lymphangitis, though all the tissue elements of the part are necessarily affected. But the variety under notice, instead of spreading simply or mainly by continuous implication of the local capillaries, follows the course of the lymphatic trunks from the seat of primary infection to the next group or groups of lymph glands, in this way resembling syphilis. Thus, a wound on the inner side of the hand or forearm

sets up specific inflammation of the lymphatics that join the supracondyloid and axillary glands. The path of transit of the poison is marked by a series of red lines which remain distinct, or become fused as the hyperæmia extends. The lymphatics, and the lymph-paths in the glands, become blocked, and thus the infective matter is to a great extent arrested in its course to the blood stream. Suppuration not uncommonly occurs in the glands, and in the cellular tissue about the obstructed vessels, as the result of the irritation of the poison there accumulated.

**Phlegmonous or cellulo-cutaneous erysipelas.**—This variety of erysipelas occurs most frequently in the upper limb. It is characterised by intense inflammation of the skin and subcutaneous cellular tissue. At first the affected part is bright red, œdematous, painful, and exceedingly tender to the touch. As the tension increases the skin assumes a shining aspect, and feels hard and brawny. Later, the diffused redness becomes varied by deep maroon-coloured patches of congestion and thrombosis, and here and there a boggiess can be detected, indicating the breaking down of the subcutaneous tissues. Or there may be distinct fluctuation, especially where the phlegmon is comparatively circumscribed. Meanwhile the skin is destroyed by a combination of purulent liquefaction and gangrene. The openings made by the surgeon, or established spontaneously, give vent to the discharge, which is very profuse and composed of pus in which shreds and flakes of sloughy cellular tissue are suspended. Unless there be a wound, the signs of putrefaction (offensive smell, and emphysematous crackling from the evolution of gases) are absent. The process is so rapid that the greater part of the integument of the fore-arm may be destroyed within a week, and the muscles and tendons laid bare, or covered only with dirty-grey sloughs. From the



paucity of vessels, and the strangulation by the high tension of those that exist, the tendons frequently necrose.

As the cellular tissue melts away more quickly than the skin, the latter becomes undermined for a considerable distance, and the finger passed beneath it meets with no obstruction, or only from some isolated fibrous bands that carry the blood-vessels from the deeper parts to the skin. These vessels are often plugged with clots, and this hastens the death of the structures supplied. Care should be taken not to break them through, as profuse, dangerous, and even fatal hæmorrhage might be caused thereby, the more so if the cellulitis has reached some very extensive cellular tract, as the axilla or subpectoral space. After the surface is broken, decomposition of the fluids and dead tissues is set up, and then the danger of septic intoxication and pyæmia is very great.

The inflammation seems in some cases to start in the sheaths of the tendons (phlegmonous tenosynovitis), constituting one form of whitlow. The lymphatic glands are not always enlarged. The general symptoms are those of intense fever; the temperature reaches  $105^{\circ}$  Fahr.; there is complete anorexia and great nervous exhaustion; the tongue, at first moist and thickly-coated, tends to become dry and brown, and sordes form about the teeth. Death takes place from exhaustion, or acute blood-poisoning.

Phlegmonous erysipelas differs from the simple cutaneous form in the intensity of the inflammation, and the absence of a defined margin to the redness and swelling.

**Cellulitis.**—The pathology of cellulitis is much the same as that of phlegmonous erysipelas, but the skin is not primarily involved.

The inflammation spreads with great rapidity along the subcutaneous and intermuscular planes of

connective tissue. It is almost if not always started by a wound. The part becomes swollen and oedematous, then boggy or indistinctly fluctuating. As the cellular tissue is destroyed, the skin, cut off from its blood supply, may become gangrenous. There may be scarcely any change at the seat of inoculation, and yet the most widely-spread suppuration and sloughing of the areolar tissue beyond.

In pelvic cellulitis following operations, or parturition, the inflammation sometimes spreads to the peritoneum, but not necessarily so. I have known the serous surface to retain its lustre whilst suppuration had extended from a lithotomy wound, along the course of the ureter to the kidney. In injuries to the scalp the destruction is sometimes so extensive that the entire galea aponeurotica rides upon a bed of pus.

Cellulitis of the neck (*angina Ludovici*) may be very extensive. It usually follows some operation, or starts in inflammation of the pharynx or tonsil, set up by a wound or direct inoculation with infective matter.

The lymphatic glands are enlarged, but they do not often suppurate, although they may be found gangrenous in the sloughy debris that surrounds them.

The constitutional symptoms are strongly marked—high fever and asthenia, quickly ending in death unless relief is given by early incisions. The disease is very fatal in all its forms, but in no other more surely and rapidly than after post-mortem wounds with inoculation of the poison from diffuse inflammation, such as peritonitis.

## CHAPTER X.

## FURUNCLE, CARBUNCLE, AND MALIGNANT PUSTULE.

**Furuncles**, or boils, are localised inflammations of the skin and subcutaneous tissue. When occurring singly they probably have a purely local origin in some extrinsic irritation, or in the pent-up secretions of the cutaneous glands; hence their frequency in the axillæ and perineum. When multiple (furunculosis) they are generally considered as the expression of an unhealthy state of the blood. There can be no doubt but that they are most frequent in persons out of health, whether the cause be transient or abiding. Diabetes and chronic kidney disease predispose to them. The proximate cause seems to be an irritation of the glandular structures of the skin by some peccant material circulating in the blood.

**Morbid anatomy.**—The blood stagnates and then coagulates in the capillary areas about the sudoriparous and sebaceous glands. This entails death of the part, and inflammation around the thrombosed vessels. As the tissues melt away a bed of pus is formed, and the little isolated gangrenous plug comes away as a slough, or it liquefies. The further progress is that of a small acute abscess. More rarely the inflammation subsides without the occurrence of suppuration, and the swelling is then termed a “blind boil.” However acute the inflammation, it never spreads to the surrounding parts.

The discharge always contains micrococci—*staphylococcus pyogenes*—an organism usually found in acute suppurations. It cannot be assigned as the essential cause of boils. The same may be said of the ætiology of carbuncle.

**Carbuncle.**—A carbuncle may be defined anatomically as an aggregation of boils. It usually occurs singly, or if there be a succession of them they generally affect the same part.

The seat of election is the hard skin of the nape of the neck, and the back. This is a good instance of the part played by the tissues in the initiation of the morbid processes that affect them.

Carbuncles are most common in old people, and in those who are broken down by visceral disease; hence it is a concomitant of diabetes and kidney affections.

The minute anatomy is the same as in furuncle, but the inflammation is much more intense, and liable to spread widely from the spot primarily affected. The skin is intensely congested and hard and brawny from exudation. A number of small gangrenous areas make their appearance, at first dark, but afterwards pale, as the vessels are destroyed and the colouring matter of the blood discharged, and supuration is established around them. The islets of necrosed tissue come away, leaving apertures through which pus and shreds of slough make their escape.

Very often wide tracts of skin become gangrenous or are lost by progressive ulceration. Whilst the inflammation is subsiding in the centre, it spreads at the periphery, and this may go on until the carbuncle has attained an enormous size. There is very little tendency to destruction of the subjacent muscles and fasciæ.

The general symptoms are those of high fever, intense local pain, and considerable depression of the vital powers.

The post-mortem appearances are such as would be found after any severe inflammation. If there be evidence of septic infection and metastases, it

is indicative of pyæmia grafted upon the primary disease.

**Malignant facial carbuncle.**—This is a disease which, unlike ordinary carbuncle, has a special tendency to attack young adults. It has been confounded with malignant pustule (*q.v.*), but it differs from it in its being marked by rapidly-progressive gangrene and purulent infiltration, in the greater liability to extensive thrombosis of the veins, and in the absence of inoculation from a known specific source.

The local appearances much more closely resemble those of acute farcy, but the secondary specific metastases in the skin (farcy-buds) are wanting, and there is no history of communication of the disease by glandered horses.

Malignant carbuncle usually commences in the lip, but Billroth records a case where it began in a scalp-wound, and Paget one in the back.

The skin and subjacent structures are greatly swollen and congested, the vessels are thrombosed, and this, together with acute exudation and decomposition, destroys the tissues with marvellous rapidity. The face is so disfigured that the features are unrecognisable. The veins, which are filled with clots, mark the course of suppurative tracts.

The lymphatic glands are swollen.

There are all the signs of virulent blood-poisoning. The disease is extremely fatal, and rarely lasts longer than a few days.

**Malignant pustule.**—Synonyms, “contagious carbuncle,” “anthrax.” In the sheep, horse, ox, etc., it is known as “splenic fever,” “joint murrain,” “quarter evil,” and “the blood.” Malignant pustule is an acute specific fever, communicated to man by inoculation with the virus of splenic fever of cattle, etc. The contagion consists of a microscopical



organism, the bacillus anthracis of Cohn, the bacteridium of Davaine (Fig. 2).

The chief immediate sources of contagion are the carcasses, hides, wool, and hair of animals that have suffered from the disease; hence it is generally met with in slaughterers, tanners, wool-sorters, etc

The term "malignant pustule" is a misnomer, for suppuration is conspicuous by its absence. The "pustule" first appears as a bright red papule; upon this a vesicle forms and bursts. The central part is then converted into a black eschar with brawny œdematous base, which becomes surrounded with a crop of secondary vesicles. It is most common on the face and hands. It spreads deeply rather than superficially, in this way differing from carbuncle.

The blood coagulates in the capillaries from accumulation of the bacillus rods, and compression from without of the exudation; hence the cessation of the circulation, and the gangrene.

The surrounding tissues are congested and œdematous. The size of the central blackened eschar depends upon the duration of the disease. It may reach the size of a shilling (Greenfield). The lymph-glands of the part are generally enlarged.

Besides their mechanical effect of blocking the vessels, the bacilli, either directly, or indirectly through the products of fermentation which they give rise to, induce symptoms of toxæmia.

These symptoms vary in kind and degree. The temperature may rise to 104° F. or even higher, or it may be subnormal. The general blood-poisoning is not always proportionate to the intensity of the local lesion. In severe cases the condition is that described by Murchison as the "typhoid state"—mental depression, muttering delirium, brown tongue, muscular weakness, etc.

*Post-mortem.*—The blood is found more or less



fluid, dark, and sometimes tarry. The intima of the vessels is stained with hæmoglobin set free by destruction of the red corpuscles during life. There are local congestions, ecchymoses, and sometimes more diffuse extravasations, and more rarely wedge-shaped infarctions, in different parts of the body. The lungs are congested, as is also the mucous membrane of the alimentary canal. There are often subserous and submucous petechiæ.

**History of the micro-organism.** — The bacillus anthracis consists of *rods* varying from  $\frac{1}{2500}$ th to  $\frac{1}{250}$ th of an inch in length. They are motionless, and for the most part straight, or slightly curved. The central protoplasm is contained in denser casing. The rods multiply by fission. They also give rise to *spores* by differentiation of the protoplasm, and these are set free by splitting or solution of the encasing substance. The spores grow into rods, and thus the cycle of development is completed (Fig. 2).

The spores are very “tenacious of life;” they survive desiccation and subjection to very high temperature (100° C.). The rods are destroyed by putrid decomposition, and by exposure to much lower heat than suffices to kill the spores.

The bacilli can be cultivated artificially in nitrogenous fluids, in the presence of oxygen. As their development advances their virulence increases. The disease can be communicated from animal to animal *ad infinitum*, so the process is a truly infective one.

#### **Other forms of the disease.**

1. **Malignant anthrax œdema.**—Firm gelatinous œdema is met with in various parts, especially the eyelids. The typical pustule is absent.

2. **Internal anthrax.**—There is no primary lesion of the skin. The poison is absorbed by the respiratory or alimentary mucous membrane. The post-mortem appearances may be simply those of

acute blood-poisoning—anthracæmia. More commonly there are gross changes in the pulmonary and alimentary tracts in the form of congestion, thrombosis, hæmorrhage, and inflammation. There is often brawny œdema of the cellular tissue of the neck, and enlargement of the cervical, mediastinal, and mesenteric glands.

## CHAPTER XI.

## CELL MULTIPLICATION.

**Cell multiplication.**—There are three ways in which cells multiply : (1) by primary segmentation or division ; (2) by gemmation or budding ; and (3) by endogenous formation.

*Primary segmentation.*—It was formerly held that the nucleoli first underwent constriction and division, that similar changes followed in the nucleus, to be again repeated in the protoplasmic cell mass.

The process of cleavage of the nucleus was believed to be “direct” ; that is to say it took place without any recognisable structural alteration of the constituent nuclear substance. The researches of Flemming and Strasburger show, however, that this view is not correct. According to the former observer, the nucleoli do not divide at all, but when division of the nucleus is imminent they dissolve. To understand the series of metamorphoses involved in *nuclear division* it is necessary to bear in mind the minute anatomy of the nucleus in its quiescent or resting state. In the first place there is a limiting membrane enclosing a dense, highly refractive substance and a colourless intermediate juice. The nuclear substance contains nucleoli, granules, and filaments, which are often arranged in a retiform manner.

When the cells multiply the nucleoli disappear ; the nuclear substance proper assumes the form of a network of interlacing threads, the so-called “coil form” ; the filaments increase in thickness, become less convoluted (the “wreath form”) ; then an “aster” or star-like arrangement is obtained, the rays of which divide lengthwise ; next the filaments collect into two

polar groups of "basket form," with an intermediate transparent equatorial plate (Strasburger's cell-plate); the "basket form" passes to the "stellate"; the stellate to the "wreath"; the wreath to the "coil"; the coil to the "retiform"; and thus the cycle of

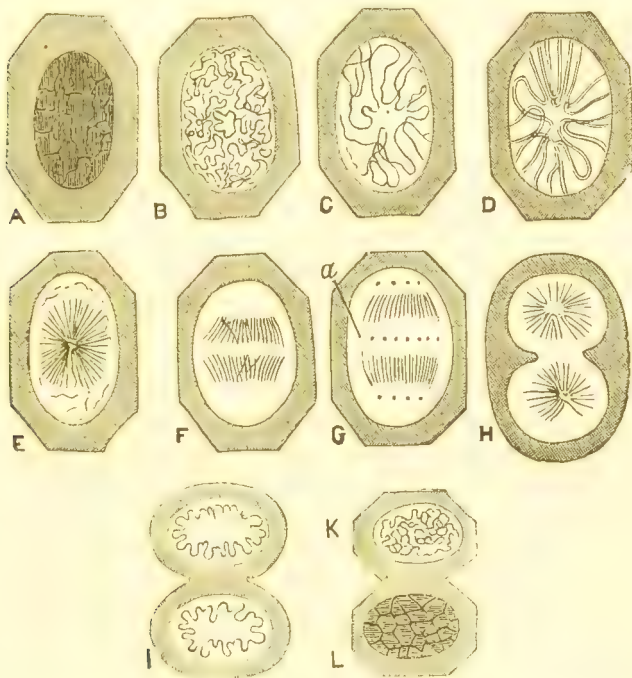


Fig. 6.—Indirect Cell Division.

A, Cell with resting nucleus; n, coil form of mother nucleus; c, wreath form showing the central and peripheral loops, some of them broken through; n, star form with free rays, showing the filaments in the act of splitting; E, fine-rayed star form; F, nucleus with equatorial plate, division into polar segments; G, half-barrel or half-spindle form, a Strasburger's equatorial cell-plate; H, star form of daughter nucleus; I, wreath form; K, coil form; L, resting nucleus with network. (From Ziegler's "General Pathological Anatomy.")

transformations (so far as the nucleus is concerned) is completed. Meanwhile, the cell mass contracts between the "asters" until the bond which unites them is severed (Fig. 6).

This subdivision is called "indirect," inasmuch as it essentially depends upon the peculiar behaviour of the nuclear filaments above described.

*Gemmation* is very like to "indirect division." The only difference is that a bud-like process of the cell protoplasm is thrust out previous to the segmentation of the nucleus.

This process receives the daughter nucleus and then detaches itself from the parent cell.

*Endogenous formation.*—It has been already stated that multinucleated giant cells originate in a continued multiplication of nuclei, whilst the cleavage of the surrounding protoplasm is arrested.

In endogenous proliferation some of the protoplasm is collected about the several nuclei, and a clear space is left between the new cells and the remaining protoplasm of the mother cell. The whole is called a "brood cell."

This method of multiplication is seen in inflamed mucous membranes and cartilage, and in the granulations of healing ulcers.

## CHAPTER XII.

## HYPERTROPHY.

HYPERTROPHY may be simple or numerical, according as the increase in bulk is due to a simple enlargement of pre-existing elements or an addition to their number. Usually the two are combined.

Hypertrophies stand, so to speak, between inflammatory enlargements and the new formations; but were an attempt made to define sharply and separate them from these groups on strict pathological grounds, a number of cases which by common consent are regarded as hypertrophies would have to be excluded; *e.g.* corns and the bursæ found beneath them, thickened bones, enlarged hairs and papillæ around chronic ulcers, enlarged prostate, etc.

Hypertrophies are essentially homoplastic; tumours are frequently heteroplastic.

In hypertrophy proper the tissue is well organised, and there is a corresponding increase in vital activity, whilst in inflammation there is a lowering of histological type and a decrease of functional power. As a rule, the increment of nutrition is preceded by a call for it, "*ubi stimulus ibi affluxus*;" but now and again this is reversed.

**Hypertrophies classified.**—(1) Spontaneous; (2) compensatory; (3) irritative.

The **spontaneous** comprise certain developmental and anomalous forms.

*In developmental hypertrophy* there is an irregular evolution as regards the mode and extent of growth.

It is either congenital, as when a child is born with an enlarged hand or foot; or it comes on later in life, at the time of a fresh phase of developmental



activity; *e.g.* a general hypertrophy of the breast about the age of puberty and early womanhood.

Enlargement of the prostate in old men may be cited as an *anomalous* case, there being no satisfactory explanation of its cause; and the same may be said of enlargements of the thyroid, thymus, and tonsils.

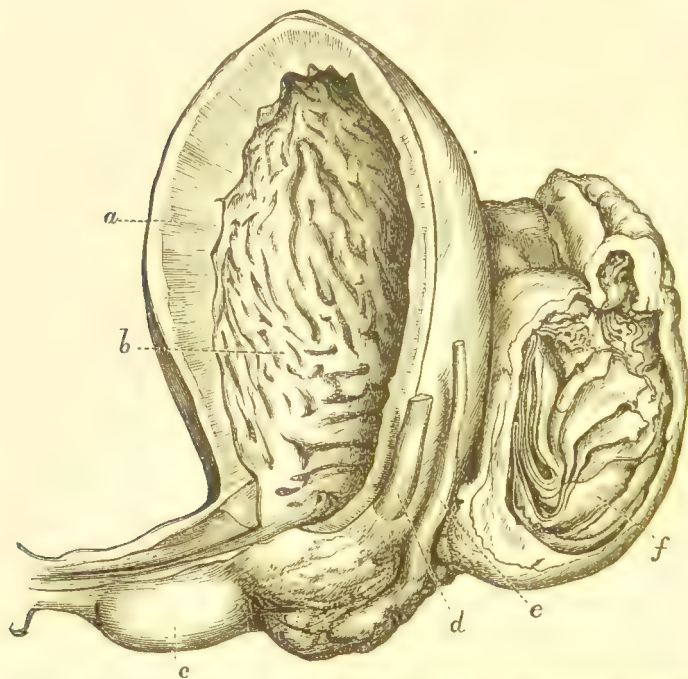


Fig. 7.—Hypertrophy of Bladder from Hydatid Cyst in the Pelvis. The parasite during its growth irritated the muscular coat of the bladder. The inflammatory thickening at the base of the viscus caused a certain amount of obstruction.

*a*, Wall of bladder seen in section; *b*, inner surface coarsely fasciculated; *c*, prostate gland; *d*, ureter; *e*, vas deferens; *f*, hydatid cyst. The essential cysts are collapsed and folded up within the adventitious cyst. (Reduced one half.)

**Compensatory hypertrophies** are for the purpose of overcoming increased resistance, or for substituted action. Thus the muscular coat of the bladder

increases in bulk that it may cope with the extra load thrown upon it by a stricture of the urethra or an enlarged prostate (Fig. 7). It would appear, however, that in some instances hypertrophy is dependent simply upon increased exercise of healthy action, such as is found in the irritable bladders of children, where there is no hindrance to the escape of urine. The heart, gall-bladder, intestine, and ureters furnish examples of hypertrophy from obstruction *a fronte*. Now, since a continuous or frequently recurring stimulus is necessary to this form of hypertrophy, it is evident why the involuntary muscles should exhibit it more than the voluntary. Voluntary muscles (*e.g.* of the limbs) increase in bulk up to a certain point when called into unwonted action, but beyond this they tire, and then the repair during the shortened intervals of rest is not equal to the loss sustained by over-exercise. But where there is naturally more or less continuous action, as in the involuntary muscles, there is scarcely any limit to hypertrophy so long as there is no interference with the supply of properly oxygenated blood. Thus there is nothing to show that thickening of the muscular coat of the intestine above a stricture ceases of itself whilst the resistance continues to increase.

The same holds good in all other instances where the natural stimulus is intermittent; *e.g.* in the glandular organs, such as the testicles.

If one kidney be crippled or destroyed by disease, the other undergoes considerable enlargement. There is undoubtedly an increase in the size of the tubules, but whether new ones are formed is a disputed point. Paget asserts there are.

If the tibia be short from arrested growth, from injury or disease of the epiphysis, the length of the limb may be preserved partially or entirely by an elongation of the femur.

Allied to the forms are the *associated hypertrophies*, where the changes follow others in parts intimately connected with them ; *e.g.* eccentric hypertrophy of the bones of the skull, when the contents of the latter are increased, and concentric when they are diminished. The thickening in these cases is best seen at the seat of the original centres of ossification.

The **irritative hypertrophies** are caused by intermittent pressure, for continuous pressure leads to atrophy. Thus the papillæ become enlarged, and the epidermis is thickened in the form of corns and callosities ; and beneath these bursæ may develop which diffuse the abnormal pressure from a tight shoe, or that misdirected in club foot.

In all cases of hypertrophy there is an increased supply of blood, and the more active the renewal the more nearly do the newly-formed elements conform to the physiological type. Thus in the pregnant uterus the circulation is very active, and there is a rapid and perfect reproduction of involuntary muscular fibres, and also an enlargement of pre-existing fibres from excessive nutrition. As the necessity for the hypertrophy is withdrawn after delivery, atrophy sets in ; the firm contraction of the muscular walls itself diminishing the blood supply. But let some mechanical cause interfere with the natural involution of the uterus, and the organ will remain enlarged for an indefinite period, not from mere failure of the atrophic change in the muscular fibres, but from the overgrowth of connective tissue (areolar hyperplasia), the result of mechanical congestion.

These cases show how a chronic inflammatory enlargement may overlap and simulate a true physiological hypertrophy ; and there can be no doubt but that the latter may be maintained to some extent by the increment of nutrition set up by the local mechanical hyperæmia.

When the original stimulus, instead of being physiological and simply calling for increased functional power, is an artificial one (*e.g.* friction against the skin, or the irritation of the hair follicles and papillæ and subjacent bone in a case of chronic ulcer), the plastic exudation is not all used up in fashioning tissues after the likeness of the normal histological elements of the part. Some, at least, pass no farther from the embryonic type than indurated connective tissue.

It would be a distinct gain if the word hypertrophy were reserved for cases where there is a call for increased functional activity and compensation, and the term *irritative overgrowth* employed to designate those arising from accidental stimulation, which produces true inflammatory enlargement.

## CHAPTER XIII.

## ATROPHY.

DEVELOPMENT and growth, discharge of healthy function, with maintenance and repair, and finally decline and death, make up the sum total of the life-history of all the tissues. The wasting of old age can scarcely be looked upon as evidence of disease ; but it rarely happens that the individual passes through his existence free from abnormal change. People grow old before their time ; some in one structure, others in another.

This premature agedness may show itself in blanching and loss of hair, decay of teeth, loss of the elastic tread of youth, senile atrophy of the brain, or weak and fatty heart.

All these conditions are attended with a diminution in the nutrition of the tissues whereby their proper constituents lose in bulk and function.

Atrophies are *simple or essential*, or *secondary* to some more actively destructive process. The former we see in the *arcus senilis* and degenerated arteries of the aged, the latter in wasting of a bone by pressure of an aneurism or its interstitial absorption by inflammation, new growths, rickets, or mollities ossium.

Inflammatory differs from simple atrophy in that it is often followed or accompanied by constructive or organising changes. Thus osteophytes are almost always found in the neighbourhood of joints destroyed by caries.

**Causes and varieties of atrophy.** — (1) *Natural or physiological atrophy* ; e.g. of the uterus after parturition, of the breast after lactation, of the testicles of the deer after the rutting season.

To these may be added the spontaneous withering of the thymus and the thyroid glands, and simple senile wasting.

(2) A part may waste from *want of its proper amount of functional stimulus*, as in the case of paralysed muscles, of the optic nerve from blindness, and of amputation stumps.

(The natural stimulus to the nutrition of a bone is the contraction of the muscles attached to it; and after an amputation the muscles have less work to do.)

(3) *Atrophies of nervous origin*.—The loss of nutrition may be largely influenced by disease of the nervous system.

The secondary descending degenerations of the spinal cord, consequent on lesions in its substance higher up, or in the brain; the wasting of the distal portion of a divided nerve; and the absorption of the articular ends of the bones in locomotor ataxia, are instances in point.

(4) *Partial deprivation of blood-supply*, as when, in fracture of the shaft of a long bone, the nutrient artery is torn; or the brain and heart soften from obstruction in their vessels.

Then there are numerous instances showing the result of *continuous pressure*. Absorption of the hard palate by a badly fitting obturator; erosion of the vertebræ by an aneurism or tumour (Fig. 8); changes of shape of the ends of bones in unreduced dislocations; the ball-and-socket pseudarthrosis of an ununited fracture; and, lastly, shrinking of the testicle from varicocele.

(5) *Excessive functional activity* tells its tale in the form of atrophy of overworn, jaded brains.

The waste from the great expenditure of nutritive force is not repaired for want of the natural amount of rest.

When, exceeding the ratio of general emaciation,



the heart grows smaller, to accommodate itself to a diminished amount of blood, the atrophy is called "purposive."

This is sometimes masked by a relative hypertrophy, when opposite causes are at work in the same patient. Thus the organ may retain its normal weight, though atrophied, as it were, from the wasting of phthisis or cancer, and hypertrophied from the obstruction of arterio-capillary fibrosis (Sibson).

**Modes of atrophy.**—The process may work out its effect by a simple diminution in the size, and eventual loss in number, of the tissue elements, those that remain being natural in appearance and consistence; *e.g.* the fibres of striated muscle. But more often obvious degenerative changes can be seen, the chief being those due to fatty metamorphosis and infiltration. Muscles that have lain fallow for a long time from paralysis, or from forced rest in joint disease, as a rule become pale and soft. The bones which in the latter case are the seat of interstitial absorption, have their spaces filled with fat, and readily fracture when attempts are made to break down adhesions in the articulations.

Perhaps the best example of fatty atrophy is the *arcus senilis* of the cornea, which commences in a degeneration of the stellate corpuscles. It is a condition of great pathological and clinical importance, from its frequent association with wide-spread vascular decay.

Probably some of the deaths from chloroform are caused by acute distension of the degenerated walls of the right cavities of the heart during the struggling stage rather than by the direct effect of the poison.

In addition to fatty transformation of the proper elements, the connective-tissue cells become loaded with fat from infiltration; and in this way an organ or tissue in an advanced stage of essential atrophy

may retain or exceed its normal bulk; *e.g.* the enlarged calves in pseudo-hypertrophic paralysis.

In certain structures *pigment* granules are deposited. This is particularly the case with the ganglion cells of the nerve centres; *e.g.* those in the anterior

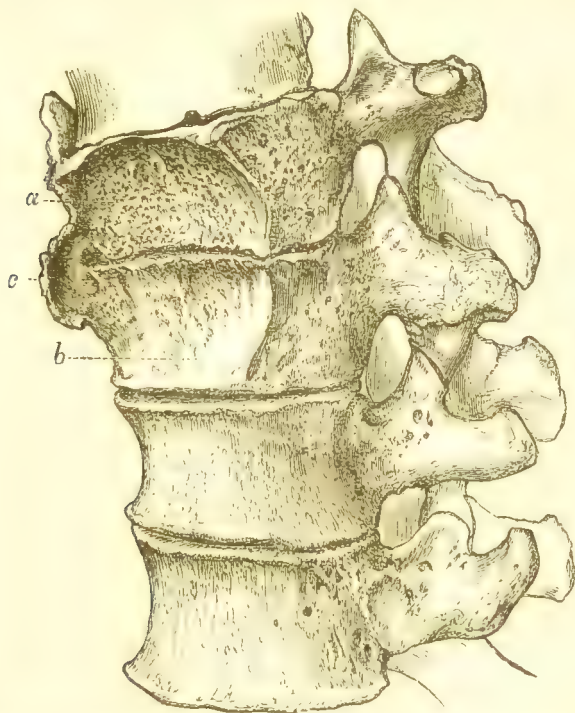


Fig. 8. —Vertebra absorbed by an Aneurism. (Reduced one-third.)

*a*, Cancellous bone forming floor of cavity; *b*, compact bone forming floor of cavity; *c*, new bone by which the vertebrae are ankylosed.

cornua of the grey matter of the spinal cord in infantile paralysis.

As nutrition fails in the muscular coat of the small and medium-sized arteries, in the rib cartilages, and in many other situations, the parts become petrified with *lime salts*.

Inflammatory deposits waste by a fatty and a *mucoid* or "liquefactive degeneration." The latter change is best observed in caries of bone. There the protoplasm of the cells becomes fluid, and lactic acid is formed (Cornil and Ranvier).

**Atrophy of bone.**—That continuous pressure causes atrophy, and intermittent pressure hypertrophy, is sometimes seen in the same preparation.

In the museum of St. Mary's Hospital there is a portion of the dorsal spine in which the vertebræ have been hollowed out by an aneurism of the descending thoracic aorta. The floor of the cavity is quite uniform, contrasting with the irregularity produced by caries or malignant disease.

On the right side these vertebræ have become anchylosed by the formation of a considerable amount of new bone; *i.e.* there is loss of substance on the left side and central portions where the pressure was constant, whereas the right margin, which was subject only to the intermittent pulsation of the aneurismal sac, is hypertrophied (Fig. 8). According to Paget, the cancellous tissue is not exposed in these cases, a layer of compact bone covering the surface. The specimen in question, however, shows in some parts the open fretwork of the interior of the vertebræ, though not so plainly as in caries or malignant disease.

The *modus operandi* of the pressure is obvious; the blood-vessels of the adjacent layer are compressed, and the bone wastes from want of its proper nutritive supply, and from absorption by granulation tissue.

The bones in old age undergo rarefaction and the earthy salts are increased; hence they easily break. Intracapsular fracture of the neck of the femur from slight force indirectly applied is an instance of this. The atrophy in these cases is eccentric. The size and shape of the bones are not necessarily altered. The medullary canal is enlarged. In edentulous jaws,

however, the alveolar margin is absorbed, and the bones are in every way smaller, because the jaws are less used for the purposes of mastication.

**Atrophy of muscle.**—The various causes of atrophy are well illustrated in the muscles, voluntary and involuntary, plain and striped.

Disuse leads to wasting of the muscular coat of the bowel below an artificial anus.

The muscles of an amputation stump gradually shrink and shorten from interstitial atrophy, and not from functional contraction. This is one reason why artificial socket-limbs are not ordered for some months after operation.

If the flaps are too short, the atrophic shortening may stretch the cicatrix over the end of the stump, and cause obstinate ulceration.

Ossification not being completed in children, the bone continues to grow, and this must be remembered as another cause of conical stump. As the growth of the diaphysis of the humerus and tibia takes place chiefly from the upper epiphysial cartilage, the deformity in question is mostly seen after amputation of the upper arm and the leg.

If muscles be kept habitually shortened, as in flexion and adduction of the thigh from hip-joint disease, they will shrink. This may offer a serious bar to the straightening of the limb, and may necessitate division of the tendons.

In acute inflammation of joints, the irritation of the articular nerves seems reflexly to impair the nutrition of the associated muscles, and to lead to a wasting out of proportion to the disuse.

Disease of the motor ganglion cells of the spinal cord in progressive muscular atrophy and in infantile paralysis causes a corresponding wasting of the muscles supplied by them.

In infantile paralysis, where the loss of power is

often sudden and complete, the subsequent wasting from disuse may to some extent be averted or removed by an artificial stimulus, such as passive movement or electricity.

When there is advanced general fatty degeneration of the muscular system, no operation should be undertaken that is not absolutely necessary, for the nutritive activity, which is at a very low ebb, would not unlikely fail to repair an extensive wound.

**Atrophy of nerves.**—In addition to what has been already said, we may cite softening of the brain from ligature of the carotid, embolism, etc., degeneration of nerves whose function has been annulled or repaired; *e.g.* in amputation stumps. According to Dickinson, the bulk of the nerve may be retained but the fibres wasted, fat and connective tissue taking their place.

## CHAPTER XIV.

## FATTY INFILTRATION—FATTY DEGENERATION.

PHYSIOLOGICALLY, fat exists in the animal tissues in two forms, firstly, in combination with albuminoid constituents, and secondly, in the free state, as granules or droplets in the cells. In like manner we find it in pathological states either as a mere infiltration, or as the result of metamorphosis of cell protoplasm.

**Fatty infiltration.**—The cells of the liver, and especially those forming the outer zone of the lobules, always contain a certain amount of fat, elaborated and stored up by the functional and nutritive activity of their protoplasm. This is notably increased during digestion. The excess is used up in the intervals of feeding to supply the requirements of the system, both for maintenance and repair.

By its combustion heat is given off, and this keeps up the normal temperature, and, being transmuted into physical force, works the complicated machinery of the body.

The villi of the intestines and the lacteals are also loaded with fat during digestion.

Under ordinary conditions the balance between waste and supply is maintained; but this may be lost on either side. Thus, if the amount of food be insufficient, fat quickly disappears from the cells, and the animal emaciates. On the other hand, obesity is the consequence of overfeeding and inaction. (The question of demand and supply is too often lost sight of in the treatment of patients taken from active life and suddenly confined to bed. Apart from indiscretion in diet, such a condition is sufficient in itself to



cause furred tongue, indigestion, headache, and a rise of one or two degrees in the body temperature. Hence the advantage of complete rest and modified regimen for some days prior to the performance of any severe operation.)

It is impossible to say where a physiological becomes a pathological adiposity, but certain it is that the secretory function of an organ may be strained by excess of work, just as much as a muscle used beyond certain limits will tire and waste.

There can be no doubt but that overfeeding long continued is a cause of organic disease of the liver, and this again of the whole of the digestive system. The connection between the "pleasures of the table" and hæmorrhoids is well known.

Although fatty degeneration and infiltration are quite distinct processes, they are not uncommonly associated; *e.g.* in "fatty heart" the muscular fibres have undergone a retrograde change, their constituent fat having been liberated. At the same time a considerable accumulation of fat in the cells of the connective tissue beneath the pericardium and between the muscular fasciculi is far from rare. Again, in certain *paralytic states*, and especially in "pseudo-hypertrophic paralysis," the muscles are enlarged in the gross in spite of the wasting from fatty atrophy of their fibres. There is an infiltration within the sarcolemma and between the muscular bundles.

The mere *disuse of a part* is often followed by a marked fatty infiltration; thus, if a limb be kept fixed for a long time, the bones become rarefied from simple atrophy, and the cells in the enlarged cancellous spaces filled with fat. A similar change is observed in the muscles. The bones are rendered more fragile, and so easily break on applying force to overcome stiffness in the joints. The muscles are less contractile,

and hence amputation flaps retract but little, and their state of lowered vitality does not conduce to rapid healing.

In *phthisis* a noteworthy feature is enlargement of the liver from fatty infiltration. The reason of this is not clear. It may be from the impaired function of the lungs, but it must not be forgotten that high temperature leads to a general absorption of adipose tissue, and it may be that the blood thus overloaded is relieved of its incubus by the liver.

Finally, although fatty infiltration may mechanically impede the contraction of muscle, and hamper the secretory function of glands such as the liver, it is not incompatible with the continued life and activity of the tissue elements affected.

**Fatty degeneration.**—Here there is a true metamorphosis of the cell protoplasm. By some it is alleged that albuminoid bodies are converted directly into fat, but it is more probable that fat normally exists as a constituent of cell composition in intimate combination with nitrogenous substances.

As the result of chemical decomposition, the constituent fat is set free, and the minute particles give a granular appearance to the cells.

Physiologically, fatty degeneration occurs in the mamma, and the sebaceous and ceruminous glands. The functional activity of the cells involves an end to their vitality. They become loaded with fat, then disintegrate. In the meantime new cells replace those that have disappeared, and so the balance of nutrition of the secreting structures is maintained as long as sufficient pabulum is afforded, and the secretion is carried on within natural limits. Over-action leads to exhaustion and wasting.

During the involution of the uterus after parturition the hypertrophied muscular fibres undergo retrograde fatty metamorphosis prior to absorption ;

and the same happens in the corpus luteum of the ovary.

In old age fatty degeneration plays an important part in the natural decay of the tissues. Of such nature is the *arcus senilis* of the cornea. As a rule, this first appears in the upper segment as a dull, whitish crescent, at a short distance from the sclero-corneal junction. A second crescent is formed below, and these two extending, the circle is completed. It is of little clinical importance in itself, but it serves as an index to similar changes in other parts of the body, and particularly the vascular system. It points to the likelihood of fatty degeneration of the heart and arteries, and thus to the dangers of syncope and apoplexy.

It is an interesting fact that in an eye that has been the seat of organic disease, even though all visible signs have disappeared, the fatty degeneration of the corneal corpuscles will come on earlier than in the previously sound eye. This is a good example of lowered vitality constituting a "*locus resistantie minoris*."

Another instance of fatty degeneration and natural decay of tissue elements is seen in the epithelioid cells lining the large arteries, which we have observed in children only three years of age (Fig. 9, A). This, and the senile changes above referred to, can scarcely be regarded as signs of disease. (*Vide Atrophy*, pages 114-120.)

At the same time, these fatty degenerations from old age are of the greatest moment in the consideration of surgical pathology, bearing so directly as they do upon the prognosis of disease, and the repair of wounds. They would count in the scale against the performance of operations that are not absolutely necessary.

**Causes of fatty degeneration.**—The circumstances that conduce to fatty degeneration are,

(1) Inherent disposition to decay ; (2) defective vascular supply ; (3) rapid growth ; (4) high temperature ; (5) the action of poisons.

There may be a combination of causes, as in inflammation and the acute fevers, especially those of infective origin :

(1) This was discussed when speaking of true physiological degenerations, and the allied changes dependent on old age. To these may be added cataract of the crystalline lens, and notably the fluid and soft varieties.

(2) There are several ways in which *deficient supply of blood* entails fatty degeneration. It is seen in its simplest form in diseases that diminish the elasticity and lessen the calibre of the arteries, either by a primary calcification of the middle coat, or thickening and irregularity from chronic and sub-acute arteritis. This is well illustrated in the *heart*. At times the orifices of the coronary arteries are greatly narrowed from atheroma of the first part of the aorta ; more frequently the walls of the arteries themselves are extensively affected as well. Anæmia (in its physiological sense) over wide tracts follows obstruction from embolism and thrombosis and their consequent infarctions, the extent of it depending upon the freedom of the collateral circulation. In the brain, where this is very limited, softening is sure to ensue if a vessel of considerable size be blocked. Although three varieties of *cerebral softening* are described (*red, yellow, and white*), there are two factors only that contribute to the result : firstly, the suddenness and completeness of arrest of the blood stream ; and secondly, by implication, the amount of blood in the part. In all three there is fatty degeneration of the brain substance and of the products of exudation. If a good-sized vessel be plugged by an embolus, the venous reflux and capillary stagnation and rupture

reach their limit. The colouring matter of the disintegrating infarct mingles with the fatty débris, and a semi-diffuent red pulp obtains. If, on the other hand, the arrest be gradual, there will be sufficient force to drive the blood through the obstructed artery and its capillaries and veins beyond ; but the current will be slow and small, and inadequate to the maintenance of the tissue it supplies, which will consequently waste from fatty atrophy. Between the red and the white softening stands the yellow, but it represents only one shade of many passing from red to white.

At times a steady degeneration goes on for a period, giving rise to a white softening, and this is subsequently modified by capillary rupture at the margin.

Fatty degeneration of the heart and atheroma of the cerebral arteries are the precursors of white softening ; sudden occlusion by embolism or thrombosis, of red.

Cerebral softening is never sharply defined, but passes gradually into the surrounding healthy brain substance.

Under the microscope there will be observed, in variable quantity according to the kind of softening, blood corpuscles, pigment granules and crystals, large compound granule cells, free fat particles, crystals of fatty acids, and cholesterine, and, if the process have been rapid, shreds or sloughs of brain tissue.

Fatty degeneration is a constant feature in all *inflammatory exudations*. It is chiefly due to the insufficiency of blood supply from the tension on the capillaries ; but the increased temperature of the blood in general, and of the inflamed part in particular, and probably also the deleterious effect of the chemical products of decomposition of the exudation, have much to do with it. It involves not only the pus



corpuscles, but the walls of the blood-vessels, and, in fact, all the elements that make up the inflamed tissue.

(3) In rapidly growing *tumours*, such as cancers and sarcomata, fatty degeneration often reaches such a degree that large tracts break down into a diffuent pulp, constituting "softening cysts." There are two reasons for this extensive fatty change: 1st, the formation of blood-vessels cannot keep pace with the growth of the tumour, and, from the softness of the latter, capillary ruptures are common; and 2nd, the vital activity of the cell is expended in their multiplication, to the exclusion of a high development, hence, being unstable, they wither and die.

(4) *Hyperpyrexia* is a powerful cause of fatty degeneration, and cannot well be overrated, whether it be considered from a pathological standpoint, or in its practical application to the treatment of disease. Post mortem all the organs are found softened, and break down readily under the finger. They feel greasy, and are of a dirty yellow colour.

The rapid transformation of protoplasm into fat explains the danger of fatal syncope from intrinsic failure of the heart.

The beneficial effect of bringing down the temperature by the cold bath, or large doses of certain drugs, such as quinine, is very marked. The heart acts more powerfully, and the pulse becomes slower, and headache and delirium are decidedly checked.

(5) The *action of poisons*.—Poisons causing fatty degeneration naturally fall into two groups:

(a) The *materies morbi* of infective fevers, including the acute specifics, and septicæmia.

(b) Certain mineral poisons. The chief of these is phosphorus, then come antimony, arsenic, and mercury.

How they act is uncertain. It may be that they diminish or destroy the vitality of the tissues, and that these, no longer restrained by the conditions of



life, fall a prey to chemical decomposition, of which fat is the chief product. That death is the cause, and not the consequence, of fatty degeneration, is shown by the fact that when dead bodies are allowed to

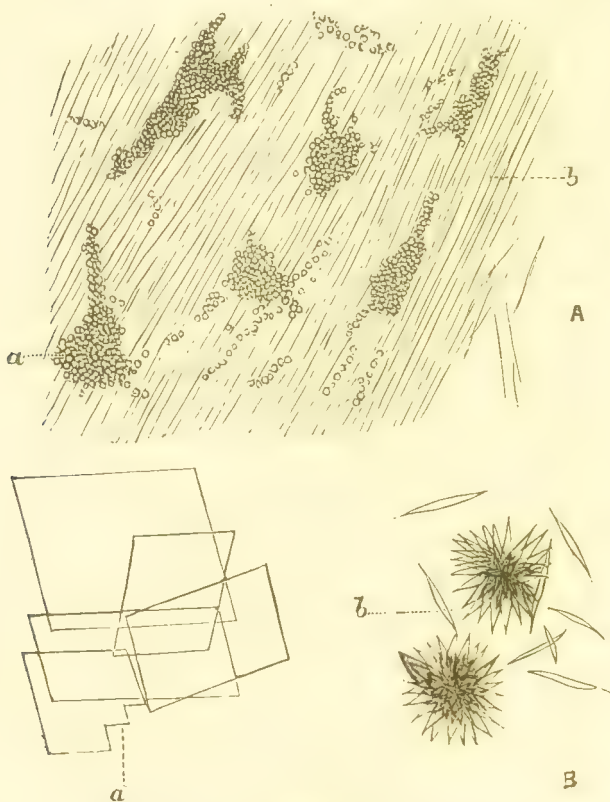


Fig. 9.

A, Fatty degeneration of the tunica interna in a flake of this membrane. In the midst of the fibrillated tissue, *b*, are seen masses of fat granules, *a*, resulting from the fatty degeneration of the flat ramifying cells of this layer ( $\times 200$ ).  
 B, Secondary products of fatty degeneration. *a*, plates of cholesterol; *b*, crystals of stearic acid (Cornil and Ranvier).

decompose slowly, under the influence of moisture, ordinary putrefaction is arrested, and the tissues are converted into a fatty body termed adipocere.

**Microscopy and chemistry of fatty degeneration.**—Fat granules appear first around the nuclei of the cells. This can be easily observed in muscle, where the nuclei are large and elongated. The fat particles are here deposited in parallel streaks. They are very numerous, dark, and highly refractive. The smaller ones merge, but the cell is not distended by a large drop, as in fatty infiltration. After the cells break up the granules are dispersed, and are then more readily absorbed. Absorption is extensive and very constant, and hence it is not surprising that fatty degeneration is the principal mode of atrophy. If the fat be in large amount, or long retained, it breaks up into the fatty acids and cholesterine. Stearic acid crystals are rhomboidal, acicular, and stellate. Cholesterine appears as plates, with pieces chipped out of the corners (Fig. 9, *b*).

Fat granules are dissolved by ether and strong solution of caustic alkali; they are turned back by osmic acid, but do not take the staining from logwood or carmine. If iodine be added to cholesterine, and then strong sulphuric acid, a blue colour is obtained. Sulphuric acid alone gives a deep red colour.

## CHAPTER XV.

## MUCOID AND COLLOID DEGENERATION.

THESE changes are very closely allied, and are found in similar circumstances. It is difficult, without the aid of chemical reagents, to distinguish between them.

**Mucoid degeneration.**—This alteration in the composition of the tissues does not appear to depend, like fatty degeneration, upon a mere diminution in nutritive supply, but to be an essential factor in the life-history of many new growths and other morbid products.

*Physiologically*, mucoid tissue is widely distributed in the fœtus, where it marks the transitional stage from embryonic to more fully developed structures, and particularly connective tissue. In the mucous membranes the transformation of the protoplasm of the epithelial cells into mucin constitutes the natural secretion.

In the main, the cells are destroyed by the process, and are replaced by others formed beneath them; but it seems probable that a dehiscence or discharge of their contents may occur without a necessary loss of vitality. Mucous tissue is permanent in the Whartonian jelly of the umbilical cord, and in the vitreous humour of the eye.

**Pathogeny.**—In the majority of cases this form of degeneration is found either in tissues, where it is a physiological constant, or in those that are but little removed from the embryonic type. In inflammation of the mucous membranes there is an exaggerated secretion. We see evidence of this in the flux of nasal catarrh, and in the ropy mucus from an inflamed bladder.

It may be stated that anything which tends to render the vitality of the tissues unstable tends also to the formation of mucin in the cells and intercellular substance. Hence it is far from rare in inflammatory products and new growths. *Tumours* that spring from the mucous membranes are commonly gelatinous; *e.g.* the simple polypi of the posterior nares. In them the mucoid transformation goes on *pari passu*

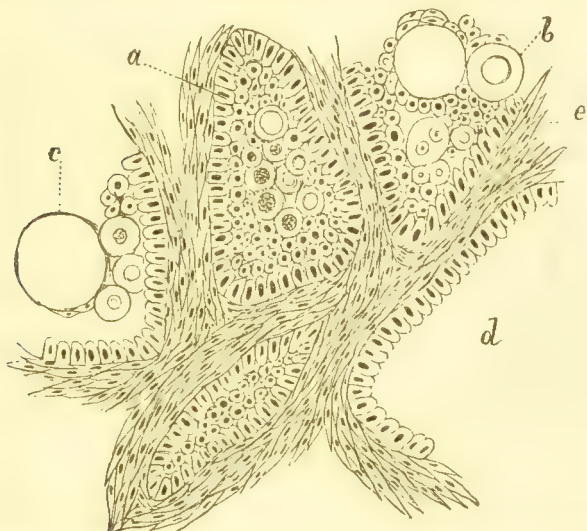


Fig. 10.—Multiple Cystic Epithelioma of Lower Jaw (Epithelial Odontome) Cyst Development in process.

At *a* the central round epithelial cells are undergoing distension from mucoid metamorphosis; at *b* and *c* this is further advanced; at *d* all the central cells of the loculus have disappeared. The peripheral columnar cells remain as an epithelial lining to the cysts, *e*, interlobular tissue, composed for the most part of spindle-shaped cells; in other parts of the growth they had developed into fibrous tissue. The tumour from which the section was taken is represented by Fig. 81.

with the increase of tissue, so that there is a uniform glistening throughout. Growths of similar structure are found springing from connective tissue in various situations.—beneath the skin and fasciæ, and in the parenchyma of organs, the parotid gland, for example.

There are some tumours in which the mucoid change is not generally diffused, but scattered in patches as a secondary degeneration and softening. By the fusion of contiguous droplets cysts are formed, the walls of which are smooth or rugged, and the contents semifluid, and either clear or turbid, colourless or stained. If coloured or turbid, it is due to admixture with granular fatty matter or blood pigment, and not to alteration in the chemical composition of mucin (Fig. 10).

Of this nature are many cystic sarcomata and enchondromata.

The costal and articular cartilages and the intervertebral discs are liable to undergo this form of degeneration. It is seen in the rib cartilages of old people, in joints affected by "white swelling," and in the spinal column, the seat of caries.

The glairy discharge and gelatinous granulations of many ulcers are explained in the same way.

The existence and general distribution of mucoid degeneration rest upon a physiological basis; but there seems to be no satisfactory explanation why gelatinous polypi should be common in the nose and rare in the rectum and bladder, nor why some growths should be riddled with mucous cysts whilst others of like structure remain free.

**Microscopy.**—The fibres of connective tissue swell up, and their outline becomes obscured and finally lost. Elastic tissue resists the change much better than the white variety. When the cells are affected, a drop of mucin appears in the protoplasm (Fig. 10, *b*). It enlarges and pushes the nucleus to one side. Eventually all signs of cell wall and nucleus disappear. In the myxomata and in the umbilical cord, the outrunners of the branched cells unite to form a delicate network, spun, as it were, through the homogeneous gelatinous matrix. (*Vide* Figs. 77*b*, 78*b*.)

The secretion from inflamed mucous membranes contains granular leucocytes, and cells of epithelial origin.

**Chemistry.**—Mucin is only found in alkaline fluids. It is precipitated by alcohol, alum, and dilute mineral and acetic acids. The precipitate redissolves in excess of mineral acid, but this is not the case with acetic. It swells up in water, but is not soluble in it.

Mucin reduces cupric sulphate. It is converted into acid-albumin by the mineral acids.

Though closely allied to albumin, it differs from it in not being precipitated by bichloride of mercury, tannin, nor by boiling; and in the absence of sulphur from its composition.

Its congeners, gelatine and chondrin, contain a small percentage of sulphur, and are precipitated by alum and mercuric chloride. Mucin may be considered as the chemical product of the retrograde metamorphosis of these bodies, a reversion to the embryonic state.

**Colloid degeneration** is closely related to mucoid. It affects chiefly the cells, but the intercellular substance is not exempt. Physiologically it is sometimes met with in the thyroid gland, even in very young children.

It is conspicuous in many cases of bronchocele. In them the change may be confined to individual glandular acini; but very commonly, by the coalescence of the latter, cysts are formed, which contain a semi-gelatinous, colourless, or yellow fluid. It gives the characteristic appearance to colloid cancer, which has a special tendency to affect the intestines, omentum, and ovaries.

Pure colloid cancer is rare in the breast, but colloid degeneration of the cells of ordinary mammary carcinoma is of common occurrence.



We have observed secondary colloid cancer of the spermatic cord in a case where the œsophagus was primarily affected.

It is found in other growths; *e.g.* sarcomata and enchondromata.

The essential structure of tumours is not altered by its presence, though it modifies their physical and chemical characters.

The colloid material, unlike mucin, is not precipitated by alcohol; in fact its solubility in this medium is the reason why the latter is not appropriate for hardening sections of tissue affected with colloid degeneration.

**Zenkerism.**—This term has been applied to a peculiar form of degeneration, allied to colloid, described by Zenker as occurring in the voluntary muscles in *typhoid fever*, notably the adductors of the thigh, the diaphragm, and abdominal recti.

It has been observed in other febrile disorders. Cornil and Ranvier met with a similar condition in the muscles around centres of inflammation and new growths. It has also been detected post mortem in muscles that had undergone injury during life. Whether it be a distinct and peculiar form of degeneration, or, as Cohnheim supposes, a modification of cadaveric coagulation of myosin, is not certain.

The fibres affected lose their contractility, and become soft. From want of support the blood capillaries may rupture, the resulting hæmorrhages causing pain and tenderness, and even swelling along the course of the muscles.

**Microscopy.**—The alteration in structure is by no means general in the same muscle. Patches appear here and there in the midst of what looks like healthy tissue. Even individual fibres can be seen lying side by side with others that have entirely escaped, reminding one of the partial distribution

of lardaceous degeneration in the muscle cells of the blood-vessels.

The fibres affected are greatly swollen. They look glistening, and have lost their natural striation. The sarcolemma is wrinkled irregularly from transverse cleavage of its contents.

Regeneration takes place by absorption of the diseased fibres and the development of new ones.

**Myxœdema.**—In this disease the connective tissue, especially of the hands, feet, and face, becomes swollen. Its constituent gelatine and chondrin undergo a chemical reversion to the embryonic condition, being converted into mucin.

There may or may not be disease of the kidneys. The disease is slowly progressive, and the patients evince a dulness of intellect and gradual diminution of bodily vigour. The ætiology is involved in obscurity. Excision of the thyroid gland has been shown to be sometimes followed by an arrest of mental and bodily development, and a tendency to myxœdema.

## CHAPTER XVI.

## PIGMENTATION—PIGMENTARY DEGENERATION.

THE term pigmentation is of wider application than pigmentary degeneration. It is true there is a tendency to the deposit of pigment in structures that are losing their vitality ; but there are many instances that do not fall under this category ; such *e.g.* are the melanotic tumours, in which the process of fixation and storage of colouring matter is as much an essential part of the life-history of these growths as the formation and arrangement of the tissue elements that compose them.

**False pigmentation.**—In the first place, a distinction should be drawn between *true* and *false* pigments, or rather, between those that are derived from the blood (primarily or secondarily) and those that are introduced from without. Of the latter the most common form is *carbon* suspended in the air in the form of minute particles. These are carried to the respiratory mucous membrane, to which they adhere. Eventually they become fixed in the pulmonary tissue, partly by their mechanical action, and partly by the agency of the protoplasm of the cells, in the same way that granules are appropriated by amcebæ from the fluids in which they live.

Not only do these particles invade the parenchyma of the lungs, but they find their way into the lymphatics, and are arrested in the bronchial and mediastinal glands. Such accidental pigmentation is well exemplified in (1) the lungs of *miners*, which are very liable to be affected with a form of fibroid phthisis, the overgrowth of connective tissue being due to the irritating action of the sharp angular pieces of carbon inhaled.

(2) *Lead workers* and others who are the subjects of chronic poisoning by the metal, show a *blue line* in the gums. Here the lead is probably deposited in combination with the albuminoid constituents of the tissues, although it is supposed by some that it takes the form of sulphide, sulphuretted hydrogen evolved from the decomposition about the teeth combining with a soluble salt of the metal carried by the capillaries.

(3) Of like nature is the occasional discoloration of the skin from long-continued internal use of silver nitrate and bismuth.

(4) From tattooing and gunpowder explosions. We have a good instance of the former in the intentional pigmentation of the cornea for the purpose of hiding the unseemly appearance of indelible scars (leucomata).

**True pigmentation** consists of the liberation of the colouring matter of the red blood corpuscles, and its fixation by the tissues in the granular or crystalline form, not as hæmoglobin, but as some derivative, the product of chemical decomposition. The pigment may be derived directly from the blood, or through the medium of some natural secretion as the *bile* or *urine*. There are certain tissues of the body of which it is a natural constituent. Such are the skin, choroid coat of the eye, liver, spleen, muscle, and nerve cells.

Physiologically, it is temporarily increased in the skin from sunburn and pregnancy; and standing midway, as it were, between physiological and pathological pigmentation is the mottling of the skin in freckles, and the melanosis of congenital moles.

In whatever tissues pigment naturally exists, there will it appear in excess in most morbid changes that affect them, even though the immediate seat of the disease become paler than normal; *e.g.* the effect of

choroiditis is often to leave whitish patches surrounded by dense black irregular zones.

Cicatrices of the skin would at first sight appear an exception to this rule, but as a matter of fact they support it, the issue mainly turning upon the point whether the rete be entirely destroyed or no, for the rete is the natural seat of the cutaneous pigment.

### **Sources of true pigmentation and staining.**

1. The *bile*.—In jaundice the conjunctiva, skin, and urine show the characteristic colour, and so do coincidental inflammatory exudations, such as pneumonic sputa. In these cases it is more a staining than real pigmentation, for the bile pigment is in solution, and is quickly reabsorbed when the cause of the jaundice is removed.

In *gall stones* pigment is always present. It varies much as to colour and amount, and somewhat as to composition.

2. The *urine*.—The colouring matter of the urine gives the "cayenne pepper" appearance to gravel, and the pink colour to deposits of urate of soda. Urinary calculi, again, have different hues indicative or suggestive of their composition; *e.g.* the brown and fawn colour of uric acid, and the yellow of cystic oxide (changing to green on long exposure). Dr. Ord has described a blue calculus composed of indigo.

3. The *blood*, however, is the chief immediate source. The pigment is either extracted and stored up by the activity of living cells without any lesion of blood-vessels, as in melanotic growths, or it is the remains of extravasation or thrombosis. In both cases the hæmoglobin as set free from the corpuscles is in solution; it afterwards undergoes chemical decomposition, and this furnishes the variation in tint seen; *e.g.* in an ordinary bruise. It does not remain diffused for long, but assumes the granular or

crystalline form. The colour of the granules may be yellow, orange, dark brown, or black, and no doubt the composition varies accordingly. There is no proof that any definite compound, which has been described under the name of *melanin*, has any real existence; the dense black pigment so termed is one of the last products of a series of chemical changes. It does not necessarily follow, however, that change of colour always implies a change in composition.

The greater part of the colouring matter of blood clots is absorbed by the vessels, and the extent of

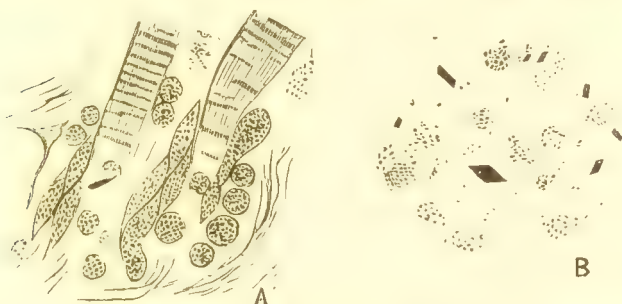


Fig. 11.—A, Melanotic Sarcoma of Muscle. B, Granular and Crystalline Pigment from a Case of old Cerebral Hæmorrhage.

absorption depends upon the freedom of the circulation; hence, in the brain (Fig. 11, B), where anastomoses are scanty, we meet with permanent deposits of pigment, and the crystalline variety is more common in this situation than elsewhere. We have seen these crystals in the fluid of hydrocele, in ovarian cysts, and in the débris of liver tissue in acute yellow atrophy. Orange-coloured granules and crystals are sometimes found in vessels blocked by coagula.

Apart from antecedent extravasation or thrombosis, pigmentation occurs

(a) As an essential feature of some *new growths* (Fig. 11, A), especially sarcoma of the choroid and



skin. We know that sarcomata are based upon the type of embryonic or developmental tissue, and that nutritive changes occur in them in greater variety than in other new formations. Cancers are much less liable to pigmentation than sarcomata. Pigmentation is not confined to malignant growths, for it is far from rare in warty excrescences of the skin. In any case, the presence of pigment marks

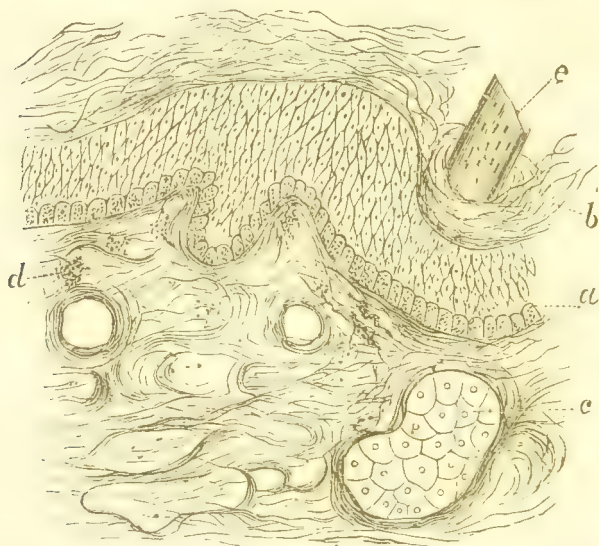


Fig. 12. —Section of Skin at Site of an old Syphilide.

*a*, Lowermost layer of rete, deeply pigmented; *b*, epidermis devoid of pigment; *c*, sebaceous gland; *d*, groups of pigment granules in thickened corium; *e*, hair

only a nutritive modification in the elements of a tumour, and has no direct reference to structural peculiarity, as suggested by the expression *melanoma*. Melanotic growths, like natural pigment, are more widely distributed in the lower animals than in the human subject. As would be expected, the granules are for the most part contained within the cells of the growth, but some are found, both scattered and

aggregated, in the intercellular substance. It is more than likely, however, that many of these collections were originally intracellular, and that they were left *in situ* after the atrophy and disappearance of the cells that held them.

(b) In long-standing *passive congestions* pigmentation is very constant, whether it has its origin in thrombosis, or capillary extravasation, or the escape of blood corpuscles without recognisable rupture of vessels. We see it around varicosed veins, and in the liver cells.

(c) In *inflammatory eruptions*, and, notably, the lymph effused in *syphilitic skin eruptions* and *syphilitic iritis*. The staining in this disease is out of proportion to the extent and duration of the vascular congestion. The difference can be explained by the supposition that the poison of syphilis is peculiarly destructive of the red blood corpuscles, and this view is supported by the fact that general anæmia is one of the signs of constitutional syphilis. It is worthy of remark that in syphilitic stains the pigment is confined to the rete and the tissue beneath, the superficial horny layer of the epidermis being quite free (Fig. 12). Either the pigment must be decomposed as the cells become cornified, fresh deposit going on meanwhile in the rete; or it is taken over by the younger cells that replace the old ones lost by desquamation. These phenomena are not confined to syphilitic stains nor to any inflammatory pigmentation of the skin; for they are found on a minor scale in the natural growth and decay of the epidermis; another proof that pathological changes are only distorted forms of physiological nutrition.

(d) The *pigmentation of the skin* around patches of leucoderma, and the bronzing of the skin and buccal mucous membrane in Addison's disease, are of uncertain origin; possibly they are instances of trophic lesions from perversion of nerve function.

## CHAPTER XVII.

## CALCAREOUS DEGENERATION.

PHYSIOLOGICALLY, calcification occurs in the development of bone, where it serves the definite purpose of giving stability to the osseous framework. But even there it marks the limit of nutritive activity of the matrix, and corresponds to the final stage of formative activity of the bone corpuscles imprisoned within the lacunæ. In caries it can be shown that granulation tissue, the anatomical product of rarefying osteitis, is of direct vascular origin, and not the result of proliferation of the bone corpuscles. Functionally, then, ossification must be considered a change to a higher state; but from a nutritive point of view it is lower than chondrification.

It may be primary, as in calcification of the tunica muscularis of the medium-sized arteries; it may be secondary and localised, as in the formation of calcareous plates in the aorta and the other large arteries affected with atheroma; or it may be secondary and widely diffused, as when several organs are infiltrated with lime salts absorbed from the osseous system.

Thus, then, calcification takes place under two widely different conditions: (*a*) from an inherent or accidental diminution in the nutrition of a tissue, without there being any evidence of excess of lime salts in the blood; (*b*) or as an act of relief, much in the same way that "chalk-stones" composed of urate of soda are formed about the joints and in the cartilages of the ear in gout, or as "de-alkalised fibrin" is deposited from the blood as a depurative infiltration in lardaceous disease. In the latter case it is found

in one or more organs. The lime salts pass through the walls of the blood-vessels in a state of solution, and are deposited outside in the interstitial tissue of the parts invaded, in the lung around the lobules, in the kidney between the tubuli uriniferi.

As before said, calcification implies tissue weakness. In *rickets*, it is true, there is excessive growth about the very parts where the cartilage capsules are undergoing calcification, but this in itself is evidence of perverted nutrition, growth, and development.

In *old age* it is rarely absent, and here it is chiefly met with in the arteries and the cartilages of the ribs and larynx.

Anything that tends to diminish the vitality of a tissue predisposes to calcification; thus we can understand why it is so frequently found in inflammatory deposits, and notably in those that are old, and the outcome of constitutional weakness, *e.g.* in tubercular glands, and phthisical nodules in the lungs.

Fatty degeneration is often found associated with it; not that one entails the other for both alike are the consequence of impaired nutrition.

Finality in the series of degenerative changes is reached in calcification, which preserves the tissue elements or their débris from further decomposition. It is a conservative process, but not necessarily by design, since in the arterial system it is a cause of embolism, thrombosis, and gangrene. If calcification sets in while the tissues retain their structural integrity, the lime salts are deposited first in the intercellular substance, and then in the protoplasm of the cells. This is especially the case in *new growths*, *e.g.* sarcoma. The cells which are hidden by the opacity of the calcareous granules can be again brought into view by dissolving out the salts with a dilute mineral acid.

Calcification may be partial or complete; in the former case it gives a roughness, and adds to the

friability of the mass, which feels something like soft mortar; in the latter the part is completely petrified and brittle. Compare the partially calcified remains of a chronic abscess with the dense plates from a large atheromatous artery.

The salts are composed for the most part of carbonate and tribasic phosphate of lime. When calcification happens in a tissue possessing characteristic chemical compounds, these will be found mingled with the salts of the earthy bases. Thus, in calcareous nodules of the kidney there are urinary constituents.

Under the microscope the calcified patches appear black by transmitted light; they are as a rule angular in outline and composed of granules. Sometimes crystals can be seen; this is the more likely when calcification has taken place whilst the affected part retained a considerable amount of moisture.

On the addition of a mineral acid the carbonate of lime is decomposed, and the carbonic acid, set free, escapes in the form of minute bubbles.

### **Calcification in the vascular system.**

1. *In the heart.*—Vegetations on the aortic and mitral valves often become calcified. The rigidity thus acquired renders them more liable to be detached, and the liability is increased if there be a secondary ulcerative endocarditis. They are a fertile source of arterial embolism, and since the stability of these detached vegetations is increased by the calcification, large arteries (*e.g.* the middle cerebral) are not uncommonly blocked.

2. *In the arteries.*—Here it occurs as a *primary* and *secondary* change. The former is seen in the medium sized arteries, such as the tibials and cerebrals. This primary calcification, one of the signs of advancing age, begins in the middle coat. The deposit commences around the nuclei; it then spreads throughout the cells which lie at right angles to the axis of the vessels, and



as the calcification is unevenly distributed, it takes the form more or less of an irregular succession of rings—*annular calcification*; and by the union of contiguous rings the artery is converted into a hollow calcareous cylinder—*tubular calcification*. Portions of the arteries are sometimes so brittle that they can readily be fractured transversely.

The process is not confined to the muscular coat; it spreads to the internal and, more rarely, external. The comparative immunity of the external coat explains why it is generally safe to apply a ligature to the artery.

The internal surface of the vessel becomes very irregular, and the lumen considerably diminished. The loss of elasticity and the increased friction, together with the diminution in vitality, favour localised or diffused thrombosis, and so become important factors in the production of senile gangrene. Other forms of degeneration, fatty and fibroid, often accompany calcification.

In the case of the cerebral arteries, if the process be slow a partial anæmia sets in, and the brain, thereby impoverished, gradually wastes, at the same time becoming firmer. Most instances of so-called senile softening are really the reverse—sclerosis. If the obstruction be great and localised, especially if the heart be fatty, and still more if the blood coagulate within the rigid vessels, portions of the brain are cut off from all nutritive supply, and consequently degenerate and soften.

*Secondary calcification.*—This is found in the aorta and other large arteries. It assumes the shape of irregular plates, and is the final stage of atheromatous disease. It affects chiefly the inner coat, for in the large vessels the muscular is but little developed. The calcareous plates protect the vessel from further change, and even serve as safeguards



against dilatation at their immediate seat. They often split away at their borders from the surrounding non-calcified portion of the vessel, and may even be bodily detached and swept along as emboli by the blood stream. They also act as foreign bodies, and cause the blood to coagulate upon them; and the thrombi thus formed are very liable to be detached, being unable to withstand the force of the current that rushes by.

3. *In the veins*.—Calcification occasionally affects the walls of the veins. *Phleboliths* are the calcified remains of venous thrombi. They are found chiefly in varicose veins of the leg, and in the prostatic plexus.

In **new growths** it is by no means rare. It has a predilection for those that arise in connection with bone, and especially sarcoma and enchondroma. The intercellular substance is first infiltrated, and then the cells. In enchondroma it is often mixed with true ossification. In some cases the distinction can only be made out with the microscope; but, as a rule, calcification is more patchy and irregular, and the absence of vessels gives to the naked-eye appearances a greater opacity. It rarely radiates from the surface of a bone, a common occurrence in ossifying subperiosteal sarcomata. Other tumours, *e.g.* ovarian fibroids and dermoid cysts, sometimes calcify.

We have met with calcification in the membranes of the brain in old people, and as the sequel of traumatic, spinal and cerebral meningitis.

Calcification of the adventitious fibrous capsules of *hydatid* cysts after the death of the parasite is not rare. We have seen such a shell a third of an inch in thickness. It may even occur between the collapsed cysts contained within the adventitious one.

## CHAPTER XVIII.

## ALBUMINOID INFILTRATION.

**Synonyms: amyloid, lardaceous, waxy.—**

In some chronic wasting diseases there is found an enlargement of the liver, spleen, kidneys, and lymphatic glands.

The causes in the order of frequency run thus:—

1. Protracted suppuration in caries and necrosis, empyema, and chronic phthisis.
2. Tertiary syphilis.
3. Cancer.
4. The cachexia produced by long residence in hot climates and exposure to malarial influence.

The above-named organs may be severally or collectively diseased. The same change is sometimes met with in the mucous membrane of the intestine.

The general characters are these: increase of size and density of the organ, a peculiar lustrous, waxy appearance on section, and a readiness with which the lardaceous substance takes the staining of certain reagents, such as iodine and methaniline violet.

With iodine it turns a deep walnut colour, changing to a blackish violet tint on the addition of sulphuric acid. It was this reaction that led Virchow to term it amyloid, though from chemical analysis it is now known not to belong to the starchy group, but to the nitrogenous, and to be allied to albuminous bodies.

With methaniline violet the diseased parts assume a blue tint, whilst the normal tissues are stained pink.

If caustic potash be added to a section stained with iodine, the colour vanishes, and it reappears on

the addition of an acid. If a portion be soaked for some time in potash, it no longer gives the reaction with the iodine even when acidulated, for the alkali dissolves out the new material. Whether the change be an infiltration or a degeneration, opinions are divided. Dr. Dickinson supports the former view, and adduces in support of it the enormous increase in bulk attained by the liver and spleen, and the early distribution of the disease in the walls of the arterioles. He believes that the material is de-alkalised fibrin, which infiltrates the walls of the vessels and exudes from them, and that it is the result of a depurative process whereby the alkalinity of the blood is diminished. In protracted suppuration there is a great loss of potash by the discharge. The albuminoid substance is deficient in potash and phosphoric acid, whereas it contains an excess of sodium chloride and earthy salts.

It must not be forgotten that there is an enormous loss of white blood corpuscles by suppuration, although they are not diminished in number in the blood; and this may be by no means an unimportant factor.

Other observers take exception to these views, both as to the exact nature of the substance and its origin. They say that other bodies, the albumins, ordinary fibrin, etc., will give a similar colour reaction with iodine, and that against its being a form of fibrin (which is a colloidal substance), is the fact that fibrin, as such, does not exist in the blood and so cannot exude from the vessels. They also argue that the special liability of certain organs to be affected, and that not in a uniform way, is opposed to the theory of mere infiltration. One fact must not be lost sight of, and that is, the tissues affected play an important part in the process, whether it be an infiltration or degeneration, and the selection may be as much on the part of *nutriendum* as *nutriens*. Those who

maintain that it is a degenerative change, ascribe it to a want of power properly to nourish certain tissues from a vitiated condition of the blood, whether it be due to a waste of alkali and corpuscles, from suppuration, or to the *materies morbi* of certain diseases, such as syphilis, cancer, and malaria.

**Anatomy.**—The change begins in and about the walls of the arterioles. The muscularis is first affected. The fibres swell and look glistening.

In the kidney the walls of the vessels of the Malpighian bodies and the vasa recta of the pyramids are notably diseased. Soon the epithelium becomes involved, and it is said exudative casts of the same substance may be found in the tubes, but these are perhaps cast-off degenerated epithelium.

In the liver the intermediate zone first suffers.

In the spleen the Malpighian bodies stand out in bold relief, looking like boiled sago grains, hence the term “sago spleen.”

In the intestine (particularly the small) the tips of the papillæ first present the waxy change.

Statistics show that this disease does not appear within three months of the commencement of suppuration.

Fatty degeneration is found associated with the lardaceous change; in fact, the albuminoid substance may itself undergo that transformation. There is not sufficient evidence to show that it organises. In rare cases it disappears, even when far enough advanced to be diagnosed during life. (*Vide* description of Fig. 40, page 290.)

The possibility of its occurrence must always be borne in mind in the treatment of chronic suppuration. Other reasons apart, it would point to amputation in preference to excision of diseased joints.

Vessels affected by the change are more permeable to fluids; hence the polyuria from affection of the

kidney, and intractable diarrhoea when the villi of the intestine are involved. The enlargement of the liver does not cause jaundice *per se*. When this is present, it depends either upon catarrh of the bile ducts, or pressure of enlarged glands in the transverse fissure (Murchison).

**Corpora amylacea.**—These are for the most part minute round or oval bodies. They have been termed amyloid on account of the colour reaction they give with iodine, or with iodine and sulphuric acid; but several authorities believe that they are nitrogenous in composition. The colour test cannot be relied upon as an absolute indication of their nature, for it led to an erroneous interpretation in the case of lardaceous disease, which, however, is widely different in its pathology from that of the bodies in question. Lardaceous disease is the outcome of a general dyscrasia, whilst the corpora amylacea are purely local phenomena, and from the frequency with which they are found apart from other morbid changes (*e.g.* in the prostate gland) they are of very little clinical import.

They are most common in the prostate, and in the central nervous system, but they have been met with in the lungs, in the mucous and serous membranes, and many other situations.

In the nervous system they are by no means rare as a sequel of chronic degenerative changes.

They are met with in the grey and white matter of the brain and cord, in the choroid plexuses of the cerebral ventricles, in the pineal and pituitary glands, and in the optic nerve and retina.

They are liable to undergo calcareous infiltration. Structurally, they consist mainly of a homogeneous material frequently arranged in concentric laminae. The lamination is probably due to successive depositions from without.

With iodine they give a blue, yellow, green, or brown colour. The extent of the three latter tints is dependent on the amount of albuminoid matter present. Iodine stains starchy substances blue, and albuminoid yellow or brown.

Sulphuric acid brings out or heightens the effect of iodine.

Amyloid bodies have not been converted into glucose, a fact which tells against their being closely affined to starch.



## CHAPTER XIX.

## T R O P H I C   L E S I O N S .

THE causes of deviations from normal nutrition must be sought (1) in the life processes of the tissues themselves ; (2) in modifications of nutritive supply, qualitative and quantitative ; and (3) in morbid states of innervation.

The tissues, like individuals, have an allotted period of healthy existence, after which they waste and die, apparently independent of vascular change or deprivation of nervous influence ; *e.g.* fatty degeneration of the corneal cells and coats of the blood-vessels. (*Vide* Atrophy, pages 114--120.)

Many lesions owe their origin to abnormal conditions of the blood and vessels. Long-standing venous congestion leads to atrophy, pigmentation, and fibroid induration, acute hyperæmia to exudations and hæmorrhages. The composition of the blood is also of great moment. On this depend the so-called cachexias of syphilis and pyæmia, and the rapid fatty softenings of protoplasm in the acute specific fevers.

But from the very intimate connection between nerve endings and the elements of the tissues, and the evidence of physiological relationship between a healthy state of the *nervous system* and the proper discharge of functions of organs, it is not surprising that any wide divergence from the normal state of the one should show itself in a corresponding modification of nutrition and function of the other.

**Trophic nerves.**—Probably there are special trophic fibres. If so, in the case of the spinal nerves they take the same course, and, in fact, are blended with the motor, sensory, and sympathetic bundles.

Meissner has shown, that if the innermost fibres of the fifth cranial nerve be divided, inflammation of the eye will result, although sensation will remain intact; whereas if only the outer fibres be cut (sensation being to a great degree lost) no such lesion is observed. In a case of inflammation of the fifth nerve that came under my notice, there was severe trifacial neuralgia, perforating ulcer of the cornea, and rapid wasting of the masseter muscle.

The **cutaneous system** presents numerous instances of the effect of perverted function of the nervous system, both as to degree and kind. Thus, *blanching, or loss of hair*, has been known to follow mental strain, severe neuralgia, and fracture of the base of the skull. Irregular *pigmentation*, as in leucoderma, is believed to be of neurotic origin. But the more frequent lesions are those where the disordered nutrition of the epidermis and true skin shows itself in the forms of glossy smoothness, eruptions, ulcerations, and transverse striation and brittleness of the nails. Here it may be remarked that these lesions are more decided when the nerves are irritated, than when completely divided. In the latter case the nutrition is defective, and there is loss of power of withstanding injuries and repairing their consequences; the process is passive rather than active.

*Vesicular eruptions* are the most common; *e.g.* *herpes zoster*, from perineuritis of the spinal nerves, and *herpes labialis* and *frontalis* in affections of the fifth cranial. The same is true of traumatic irritation. The skin supplied by the affected nerve often becomes glossy, like a polished scar, though at other times it is rougher than natural, presenting a desquamating, branny appearance, analogous to the unilateral furring of the tongue, from irritation of the gustatory nerve. *Injuries of the nerve-centres*, leading to ulceration and sloughing, as seen in the formation of *bed-sores*, from

fracture of the spine. Here the destruction of tissue is out of proportion to the extent of continued pressure on one part, from inability to shift position. Moreover, in paraplegia the sores form over the sacrum, whereas in hemiplegia there is sloughing of the buttock, on the side opposite the cerebral lesion.

*Painful irritable ulcers*, in the floor of which the terminal fibres of the nerves are probably exposed, are very intractable, and often refuse to heal until

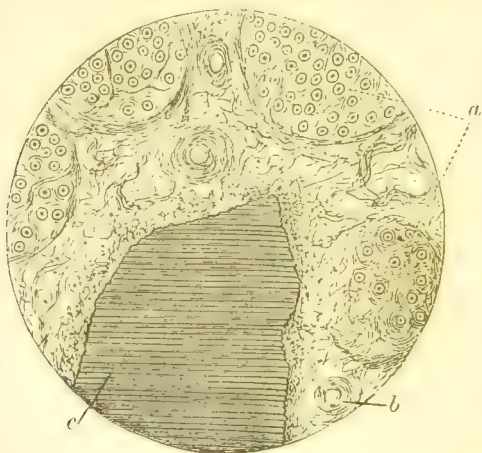


Fig. 13.—Portion of Shot embedded in Posterior Tibial Nerve.

*a*, Nerve fasciculi; *b*, blood-vessel; *c*, the foreign body. The inflammatory exudation has organised. In some fasciculi the nerve fibres are fewer than normal.

those fibres have been destroyed by caustics, or the fasciculi supplying them have been divided (Hilton).

*Profuse sweating* has been observed over the area of nerve disturbance.

Although paralysis of the vaso-motor nerves, and in some cases active vaso-motor dilatation, from stimulation (Stricker), may play a conspicuous part in the causation of *extensive œdema*, and alteration of temperature, it is probably of secondary importance as compared with affection of the trophic fibres. Such

œdema occurs in some cases of injury to the spinal cord, and also in certain diseases, *e.g.* locomotor ataxia. It may be transient or permanent.

**Perforating ulcer of the foot.**—Savory and others have pointed out that this is the consequence of nerve lesion, sometimes local, more often central. The interstitial tissue of the nerves is increased, chiefly relatively, for the nerve fibres are fewer and smaller than normal. The smaller fibres, which are probably trophic and sensory, suffer more than the larger ones.

There is abundant evidence of disordered nutrition. The *skin* is cold, frequently congested, and liable to profuse paralytic perspiration. At times, too, it is thickened, especially about the toes; and the sensibility is diminished. The *ulcer* is often seated at the base of a corn, over the ball of the great toe, or outer part of the foot, *i.e.* where the greatest pressure is brought to bear. It leads to a deep, but usually narrow, sinus, which may traverse the whole thickness of the foot, or terminate at a diseased bone. It is very intractable, and liable to return after it has healed. It has been found in anæsthetic leprosy, locomotor ataxia, caries of the spine, and in congenital deformities of the feet, which are known to be sometimes associated with disordered innervation, from defective development of the spinal centres and nerves.

Perforating ulcer of the foot may originate in *peripheral neuritis* apart from any affection of the central nervous system. The author has seen two cases following exposure to extreme cold. In one of these cases there was at first severe pain and tenderness. The cutaneous sensibility became impaired in some parts of the foot, and abolished in others; and the local circulation seriously deranged. Two years later a perforating ulcer developed.

**Connective-tissue hypertrophy.**—Short of ulceration, the soft tissues of the foot and leg may undergo hypertrophy, giving the part a coarse, clubbed, distorted look. The thickening is due to an overgrowth of an ill-developed connective tissue, similar to that seen in elephantiasis. It is more than probable that many cases looked upon as spontaneous local perversions of nutrition are really the outcome of nerve lesions.

**Muscular system.**—Paralysed muscles waste from want of their proper physiological stimulus; but in addition to this there is a more rapid atrophy, accompanied by a speedy decline in electric contractility where the trophic nerves are involved. Compare the state of the muscles of the face when palsied in an ordinary case of hemiplegia with that observed in injury to the trunk of the seventh nerve, whether directly from fracture of the base of the skull, or from the pressure of inflammatory exudation, from syphilis, or exposure to cold, or caries of the petromastoid bone.

**Osseous system.**—The repair of a fracture is liable to be imperfect, or indefinitely delayed, when the nerve or nerve-centre supplying the part is injured; thus, if the cord be crushed in the dorsal region, and a femur and humerus be broken at the same time, union will occur in the latter, perhaps not in the former.

Occasionally bone is deposited in unusual situations. Dr. Buzzard \* describes a case of locomotor ataxia, in which “a bony process appeared to occupy the position of the right rectus femoris muscle; it stretched obliquely downwards, and somewhat inwards, for a length of about nine inches.” The hip joint was affected on the same side. Fracture of the bones sometimes occurs in locomotor ataxy; this is no doubt traumatic, but it is rendered more easy by reason of

\* “Clinical Lectures on Diseases of the Nervous System,” p. 225.



atrophy from disuse (the patient being paralysed) and trophic erosion, when this is present.

The **joints**.—The joints are subject to remarkable errors of nutrition from lesions of the nerve-centres, acute or chronic, traumatic or spontaneous. Injury of the cord from fracture of the spine sometimes induces rapid inflammatory effusion.

**Charcot's disease**.—Ataxic arthropathy Charcot drew attention to extensive disorganisation of the joints in persons affected with sclerosis of the spinal cord, especially of the posterior columns—locomotor ataxy. During life the physical signs simulate those of chronic rheumatic arthritis, for which, in fact, they have been mistaken ; but there are many important differences.

In locomotor ataxia there is often considerable *effusion into the joint* ; in rheumatic arthritis little or none. This is one of the factors of dislocation, another being absorption of the articular portions of the bones.

In locomotor ataxia the onset is more sudden and the course more irregular, and the process may subside partially or entirely, whereas in rheumatic arthritis the disease is slowly progressive.

In locomotor ataxia the formation of osteophytes is the exception ; in rheumatic arthritis it is constant.

The joints are affected in the following order of frequency in locomotor ataxia : knee, shoulder, hip. Monarticular rheumatism is most commonly met with in the hip.

In rheumatic arthritis the disease usually begins in the articular cartilages, and thus creaking is an early sign. With erosion of the bones comes crepitus, which henceforth increases and is permanent, save when prevented by contracted ligaments and tendons, and bony outgrowths, or it may be complete ankylosis, which, however, is very rare. In tabetic arthropathy extensive effusion in the early stage



when the bones are not seriously affected, and the recurrence of effusion in some cases explains why crepitus is delayed, and may be afterwards obscured, when the articular cartilages are absorbed, and there is an absence of copious effusion, crepitus is manifest and indeed extremely well marked.

The absorption of the cartilages may be complete ; even the head and neck of a long bone (*e.g.* the

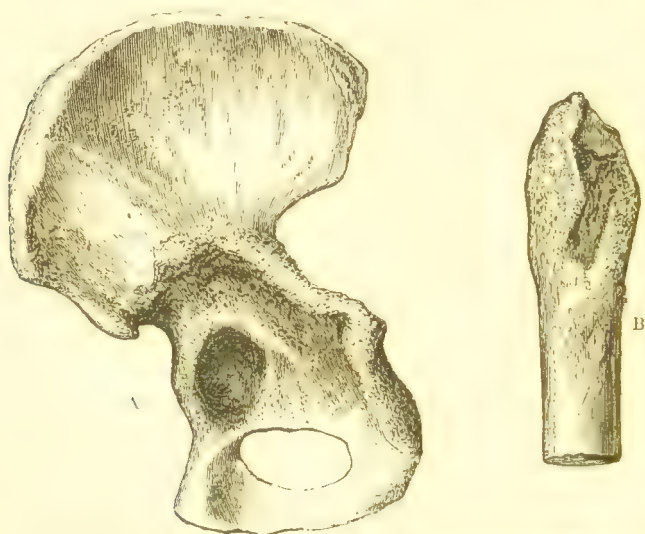


Fig. 14.—Osseous and Articular Lesions, from a case of Locomotor Ataxy.

A, Left iliac bone: erosion of acetabulum and neighbouring part; B, left femur, showing complete absorption of head (Féré).

femur) may disappear (Fig. 14). Other parts of the bone may also be eroded, *e.g.* the iliac expansion of the hip. The disease affects one or more joints. In a case described by M. Féré several were involved.

Some authorities contend that the articular lesions are not directly and entirely dependent on disordered innervation ; but that as tabetic patients—being ataxic and anæsthetic—are very liable to injury, the initial

changes in the joints are probably traumatic. Whilst admitting that the tissues concerned are defective in regenerative power, they look upon the part played by the nervous system in causing Charcot's disease as being permissive rather than directive.

**Genito-urinary system.**—Attacks of profuse hæmaturia, synchronous with lightning pains, have been observed by my colleague, Mr. Page, in a case of locomotor ataxia; the urine was quite normal in the intervals.\*

The writer has seen painless swelling of the testicles along with transient attacks of cystitis. Perhaps these phenomena were chiefly due to increased blood-capillary pressure from active vaso-motor dilation referred to by Stricker.

There can be little doubt but that trophic and vaso-motor paralyses, from crushing of the spinal cord, cause a perversion of nutrition of the urinary tract, and play an important part in the causation of cystitis and surgical kidney.

**The eye.**—In addition to what has been said of acute inflammatory destruction of the eye-ball in affections of the fifth nerve, it may be added that double descending optic neuritis is frequently seen in coarse lesions of the brain and spinal cord; *e.g.* meningitis (simple, tubercular, and syphilitic), intracranial tumours, and cerebral and spinal sclerosis. Then there is grey atrophy of the disc in locomotor ataxia, etc.

\* *Brit. Med. Journal*, vol. i. p. 772; 1883.

## CHAPTER XX.

## ACROMEGALY.

**Acromegaly** (Marie), as the name indicates, is a disease characterised by enlargement of the distal segments of the body. It may be regarded as a disorder of nutrition affecting especially the tissues of the hands, feet, face, and the cartilages of the larynx, nose, ear, and eyelids. In certain of its features it resembles myxœdema and ostitis deformans, but it has well-marked differences which justify, in our present state of knowledge, the malady as being considered *sui generis*. The principal lesion of nutrition is a slowly progressive hypertrophy. This is most marked in the hands and feet and lower part of the face. Both the hard and soft tissues are involved, but the bones suffer most. As a rule the legs, forearms, and arms enjoy immunity, but occasionally the natural bony prominences are enlarged. Usually the disease is bilaterally symmetrical. Although there is increase in the connective tissue, there is not, as in myxœdema, any excess of mucin. The skin mostly retains its normal appearance and character, but occasionally it becomes coarse, and the sebaceous glands are enlarged (Godlee).

**Changes in the head and face.**—The lower jaw becomes much enlarged so that the interdental gaps are widened; and the ability to oppose the teeth of the upper and lower jaws is lost. The lower lip is often thickened and everted. The outline of the face becomes an elongated ellipse, with the wider part below. In ostitis deformans, owing to the hypertrophy affecting chiefly the frontal and temporal bones, the face is triangular, with the base

upwards. In myxœdema the bones are not attacked, and the overgrowth of the connective tissue is more general, so that the face has a "full moon" appearance. The cartilages of the ear, nose, and eyelids often participate in the perversion of nutrition, and enlarge considerably. There is an absence of the pallor, flushing, and glossiness of the skin of the face seen in myxœdema. In both diseases there is fulness of the eyelids.

**Changes in the hands and feet.**—The hypertrophic change in the bones and soft parts may cause the hands and feet to assume enormous proportions. The creases in the skin on the dorsal and palmar aspects—especially over the joints—are deepened.

There may be certain associated enlargements of other parts. The spine frequently becomes curved, and the clavicles, ribs, patellæ, and iliac bones become hypertrophied.

Owing to enlargement of the laryngeal cartilages the voice loses its range of pitch, and may be reduced to a deep coarse monotone.

In the majority of the recorded cases the *thyroid body* was much diseased, and either enlarged or atrophied. Considering the established relation between morbid states of the thyroid and perversion of nutrition in other parts, this fact is very significant. It seems probable that the connecting pathological link is to be found in disturbed nutrition and activity of the trophic nerves.

Godlee has pointed out that malignant tumours of the thyroid gland tend to recur in bone, a further illustration of the association of morbid states of these tissues.

The *pituitary body* is in some instances much enlarged, and blindness, which may supervene as a late symptom, may thus be owing to pressure upon

the optic chiasma and tracts, causing optic neuritis and atrophy. Opacity of the cornea, which sometimes exists, would otherwise explain the loss of vision.

In acromegaly the general sensibility of the skin is preserved. The patients often suffer from spontaneous pains, especially cephalalgia. There may be deafness. There is no slowness of speech or movement as observed in myxœdema and paralysis agitans. Polydipsia, polyuria, and a cachexia of indefinite character are known concomitant conditions. In women the menopause is almost always antedated.

Acromegaly is met with between the ages of 15 and 60. The tendency is to select the periods of youth and early adult life. Ostitis deformans sets in after 40.

As further contrasting acromegaly and ostitis deformans, it may be remarked that the latter attacks chiefly the cranial bones and the long bones of the extremities; that the osseous lesions are not nearly so symmetrical as in acromegaly; that the bones of the hands and feet escape the affection; and that the soft tissues are not involved in the hypertrophy.

*Leontiasis ossea*, described by Virchow, could scarcely be confounded with acromegaly. Here the facial bones are the seat of the lesion, whilst the limbs are free from invasion by the osseous hypertrophy. (See page 275.)

In *elephantiasis* only one leg is usually affected, and the upper limbs escape. The skin and subcutaneous soft tissues are primarily involved, not the bones.

The cause of acromegaly is unknown.

## CHAPTER XXI.

## SYPHILIS.

SYPHILIS is a specific, contagious, non-miasmatic disorder, characterised by a period of *incubation* varying from one to seven weeks, usually about five weeks, by the development of an indurated sore at the seat of inoculation in the acquired disease, and by an efflorescence or rash, and generally by other inflammatory lesions. It is so strongly protective against subsequent attacks that the immunity conferred commonly lasts for the whole, or the greater part, of a lifetime. It can be communicated from parent to offspring by indirect contagion.

The local sores have been described in chapter iii., but there remain for discussion the rival theories of:—

The **unity** and **duality of syphilitic chancres**.—Some syphilographers maintain that there is but one sore, though this may manifest itself in a constitutionally non-infective or purely local form, or on the other hand give rise to general symptoms. According to this view, soft and hard chancres, so-called, are relatively pathological accidents; and the difference in the results is explained by the supposed absence or presence of the germs of syphilis. But, arguing from the analogy offered by other specific diseases, these germs must be considered as the essential elements concerned in the reproduction of the poison, and consequently in the propagation of the disease.

On ætiological grounds, no greater mark of distinction between an ulcer caused by a purely locally infective irritant and one produced by a generally infective virus can be well conceived. To meet this



objection, the supporters of the "unity" theory suppose that either the germs of syphilis are destroyed by the local ulcerative process, or that they do not find in the system a fitting soil for their development. The former hypothesis is untenable; for the poison is absorbed by the lymphatics long before any destructive action can be set up at the seat of inoculation, as shown by the fruitless attempts to prevent infection by early excision of the sore. From the nature of things, it is next to impossible to prove the latter, since no one would think of inoculating a number of persons to see if any were proof against infection. The tenacity of life possessed by the germs is probably too great for them to succumb to the action of chemical products of decomposition in the sore; for constitutional syphilis may certainly be conveyed by the secretions of most actively phagedænic chancres.

Savory considers that the evidence afforded by gonorrhœa, which is in most cases only a local affection, but which, in a few, leads to general disturbance, upholds the unity theory. But the specific nature of gonorrhœal pus is denied by some of the greatest authorities (Ricord, Lane); so the grounds for analogical inference are untrustworthy.

Attention has also been drawn to the fact that scarlatina does not always give rise to the usual constitutional symptoms. But this by no means shows that the poison is one whit the less specific, for an individual infected from such a modified source may develop all the characteristic features of the disease. It is not the absence of constitutional symptoms, but the degree of manifestation.

Again, it is said that there are many intermediate forms between soft and hard chancres. No doubt there are, but the simulation of one morbid process by another does not prove an essential relationship

between them; *e.g.* syphilitic acne and acute lichen imitate the papular state of small-pox; but each is distinct from the others. There is also a likeness between the symptoms of variola and varicella; still these diseases are none the less specific. Those who believe in the dualism of syphilitic sores explain the apparent transitional forms by the effects of local irritation, or by some peculiarity of the tissues in different individuals. By sharp or continued irritation a hard chancre may be made to suppurate, and a soft one to indurate, to a certain extent. The subsequent induration of a previously soft suppurating chancre may also be due to double inoculation at the same time, each virus producing its own results in its own appointed time.

Although there are pathological grounds for this conflict of opinions, the practical rule is never to let a patient suffering from a venereal sore pass from notice until sufficient time has elapsed for the appearance of constitutional symptoms, whether this seems likely to be the case or not.

J. Hunter maintained that gonorrhœa and syphilis were due to the same poison, from the fact that he could induce syphilitic infection by inoculation with the pus of acute "specific" urethritis; but he did not take into account the existence of urethral chancres and the infectivity of the blood and secretions of the subjects of constitutional syphilis.

**Secondary and tertiary syphilis.**—Syphilis differs from other exanthematous fevers by the long time through which the poison remains active.

The group of secondary symptoms, including the eruption, mucous tubercles, ulceration of the mouth and throat, loss of hair, induration of the lymphatic glands, iritis, and painful affections of the muscles and joints, usually pass away within the first twelve or eighteen months. Then there is a period of

quiescence which may occupy months or years, or extend through the patient's lifetime. In the latter case the disease may be considered as eradicated; in the former, the poison has lain dormant, or incapable of manifesting itself by obvious signs. After this interval it again acquires activity, and then we arrive at what is called the tertiary stage. In favour of the view of the continued potency of the virus is the uncertainty as to when the power of transmission from parent to offspring ceases, and the fact that a woman may bear an apparently healthy child between the births of two syphilitic ones, all by the same father. But many pathologists regard tertiary syphilis as the sequel, and not the direct result, of by-gone infection. They consider that the vitality of the tissues was lowered during the secondary period, and that these tissues then readily pass into a state of chronic inflammation. The non-transmissibility of the disease in the advanced tertiary stage seems to lend weight to this supposition; but against it are those cases in which the secondary symptoms pass without a break into the tertiary. Moreover, some of the tertiary lesions are anatomically characteristic of syphilis (notably, gummy tumour), whereas the "sequelæ" of other specific disorders (*e.g.* scarlet fever) are far from being so; and, even in them, there is no absolute proof that the so-called "after-results" are not, as far as causation is concerned, the direct outcome of specific irritation.

The dogmatic assertions, "syphilis once, syphilis ever," and "syphilis is a flesh and blood disease," imply not only a belief that the tertiary symptoms are specific, like the secondary, but that the patient is never freed from the original taint. The latter is opposed to the experience of many surgeons.

The type of the inflammatory processes changes considerably, for whilst the lesions of secondary

syphilis tend to spontaneous cure, those of the tertiary period are much more permanent, and show a greater liability to relapses.

Secondary syphilis, as regards tissue selection, expends the greater part of its virulence upon the cutaneous and mucous structures. Tertiary syphilis affects these parts as well, but it is also very prone to attack the viscera and the osseous and nervous systems.

Another argument advanced in support of the view that secondary syphilis is chiefly a blood disease, and tertiary an induced morbid state of the tissues, is the symmetrical disposition of the local manifestations in the former, and the irregular distribution in the latter. But whilst this holds good in the majority of cases, there are too many exceptions to warrant its general application.

The characteristic lesions of tertiary syphilis (gummata) are by some classed among the tumours, or new growths, but for no good reason. They are simply masses of inflammatory exudation, and differ from many tumours in that they cannot be enucleated, and from the malignant ones further, in that they do not generalise; moreover, they follow the course of inflammatory exudations in general in their tendency to arrive at some typical end—absorption, disintegration, caseation, etc. They are chiefly found in the skin and subcutaneous tissue; in the mucous and submucous tissues, particularly in the mouth and pharynx; in the internal organs, *e.g.* the liver, kidney, and brain; and lastly in bone. They vary in size from that of a hempseed to that of a large walnut. They may be looked on as aggregates of microscopical foci of inflammation, which, at first vascular throughout, subsequently, from degenerative and indurative changes, show three fairly distinct zones: an internal, composed of fatty and granular débris devoid of vessels;

a middle one, where the cells are round and oval and undergoing atrophy ; and an external, highly vascular and exudative. No hard and fast line is to be drawn between these zones ; they shade into one another, since they represent overlapping stages of growth and decay. The cells are embedded in a ground substance sometimes fibrillated, giving a spun-glass appearance,

but more often so thickly set with the cells as to be scarcely visible (Fig. 15).

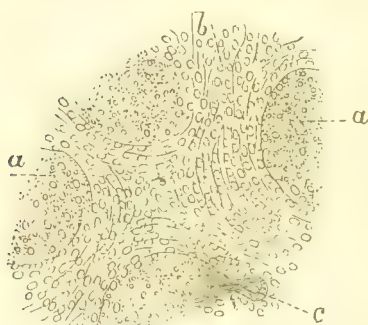


Fig. 15.—Syphilitic Gumma of the Liver.

*a*, Centres of nodes in which the cells have become granular ; *b*, periphery of the nodes ; *c*, vessel ( $\times 100$ ). (After Cornil and Ranvier.)

An old gumma appears to the naked eye as a greyish-yellow mass, surrounded by a zone of fibrous tissue. It is quite firm, and has less tendency than tubercle or infarctions to soften in the centre. In the internal organs it rarely breaks down, but the products of its degeneration, and those of the tissue

destroyed by its invasion, are slowly absorbed, deep puckered cicatrices marking the spot where it existed. In the superficial structures, where it is more exposed to irritation, a gumma very often ulcerates, causing widespread serpiginous sores, the discharge from which is at first glairy from mucoid degeneration of its substance. This may begin by superficial ulceration and gradual disintegration, or the whole mass may slough out.

The caseation is dependent firstly upon an inherent low vitality of the exudation (aplastic), and secondly upon capillary thrombosis, the consequence of syphilitic inflammation of the walls of the vessels. Amongst the amorphous *débris* may be seen stellate crystals



of stearic acid, and plates of cholesterine. Giant cells are occasionally present at the periphery, but they are not so common as in tubercle. Gummata undergo extensive mucoid change, hence the name "gummy tumour."

**Syphilitic eruptions.**—Unless rupia be regarded as essentially of syphilitic origin, all the forms of cutaneous eruptions in syphilis are modifications of non-specific varieties. The peculiarities of syphilitic eruptions are :—

1. *Polymorphism.*—The same patient may exhibit at once what is known as a mixed syphilide, *i.e.* an association of different types of cutaneous rash; *e.g.* papular, scaly, tubercular, etc. This is often seen in the secondary or exanthematous stage. It is also met with in the tertiary period. But it should be understood that this polymorphism depends rather upon the degree of local inflammation than upon any other variation in the morbid process. Thus papular passes insensibly into tubercular syphilide, the latter indicating a more extensive exudation, and therefore a greater obstruction to the capillary circulation, and liability to ulceration and interstitial destruction of tissue. Again, syphilitic psoriasis partakes more of the nature of an inflammatory exudation than of an epithelial hyperplasia.

2. *Selection of site.*—Though no part of the skin is exempt from the efflorescence or secondary roseola, the trunk, and fronts of the axillæ and elbows, are favourite situations for this form of eruption. Tubercular syphilide has a proclivity for the face, and especially the forehead—"the mark of the beast," as it has been aptly termed. Squamous syphilide in the shape of palmar and plantar psoriasis is well known. The special liability of certain parts of the skin to the action of the virus is observed in other specific fevers.



3. *Pigmentation* is more marked in syphilitic than in simple eruptions. For the pathology of this staining, see chapter xvi.

4. *Aberration from type*.—Whereas simple psoriasis has a decided tendency to affect the extensor aspect of the limbs, notably at the knees and elbows, the syphilitic form is more often observed in the flexor region.

5. *Absence of itching* is the rule, but there are many exceptions. It does not depend upon the state of tension of the nerves, for there is no certain relationship between the amount of exudation and the degree of hyperæsthesia of the skin. Either the chemical products of the inflammatory changes are but slightly irritating, or the poison of syphilis has a sedative effect upon the tissue elements of the nerves.

**Secondary and tertiary eruptions.**—The exanthem of syphilis, which is rarely or never absent, though frequently so slight as to pass unnoticed by the patient, is a *papular roseola* or *lichen*. The spots vary from the size of a pin's head to that of a split pea. The exudation is effused into the papillary layer of the skin. The rash generally makes its appearance from the fifth to the ninth week after inoculation. It is not all thrown out at once, but in an irregular series; whilst some spots are fading away others are formed. At this time the patient is somewhat feverish, the thermometer showing a rise of from one to two degrees.

As before said, the natural efflorescence is often modified by other varieties of syphilide, of which the squamous is the most common, whilst the vesicular is comparatively rare. The more severe the cutaneous lesion, the more likely it is to assume the squamous and tubercular form. Though no hard and fast line can be drawn between the secondary and tertiary manifestations, it may be said that the former are less

prone to suppurate and leave indelible cicatrices in the skin. In persons of suppurative tendency, particularly those of tubercular diathesis, destructive ulceration may commence quite early.

**Syphilitic psoriasis.** — Squamous syphilide. Small discrete patches are very frequently interspersed with lichenous papules in the primary eruption. The scales are less numerous and silvery than in the simple eruption. In some cases they form a fine circlet at the periphery of the spots, and are thinly scattered on the surface. In others the epithelial cells, embedded in fibrinous exudation, form quasi-scabs, which split here and there, giving a fissured appearance. It is not really a psoriasis, but a specific dermatitis; scaliness is common to many inflammatory diseases of the skin.

**Syphilitic lepra**, an inveterate kind of psoriasis, occurs in patients who are broken down in health ; it indicates profound constitutional disturbance. It assumes a circular or sinuous outline, so common in syphilitic affections of the skin. The surface is more or less thickly coated with exudative and desquamative products ; but as the latter predominate it presents a coarse scaly aspect.

**Plantar and palmar psoriasis** is generally of syphilitic origin. As the exudation spreads beneath the thick epidermis it raises it, and causes it to split into flakes. The heel cuticle, being very dense, resists this for some time, so that it is frequently undermined for a considerable distance, and when stripped off it leaves a moist raw surface. Cutaneous eruption of the sole is at once suggestive of syphilis and scabies, the same as ulceration between the toes. The syphilitic nature of palmar psoriasis is often confirmed by the existence of squamous patches, or superficial ulcers on the tongue. It is a late secondary or tertiary symptom.

**Tubercular syphilide.**—The term “tubercular” has no reference to the microscopical anatomy of the lesion, as in tuberculosis. It relates only to the naked-eye appearance of the eruption. The “tubercles” are firm hard nodules raised above the surface of the skin. When they form about sebaceous glands the disease is known as syphilitic acne. They have a tendency to ulcerate; but whether this is developed or not, the tissue elements are often destroyed, so that when the inflammation subsides and the exudation and softening products are absorbed, pale depressed cicatrices are left. This is all the more likely to happen when the eruption occurs in the tertiary stage, for then there is less probability of its speedy disappearance. Histologically the tubercles consist of inflammatory exudation, without any very definite disposition of the constituents. They may be considered as gummata in miniature.

**Bullous and vesicular syphilides.**—Bullous eruptions occur both in acquired and congenital syphilis. In acquired, as *rupia*; in congenital, as *pemphigus*.

*Rupia* is characterised by heaped-up incrustations on the surfaces of patches which are usually occupied by bullæ in the first instance. The bulla bursts or dries up, and the exudation and epidermis form a scab, which increases in depth by addition to its base. Meanwhile the area of inflammation widens, and consequently the scabs are somewhat conical, and as there is more or less lamination they resemble oyster-shells.

*Pemphigus*.—Children are sometimes born with a bullous eruption upon them; but most frequently it appears after birth. In the latter event it breaks out within the first few days, or, what is more usual, it shows itself about the time of the other manifestations of cutaneous syphilis; viz. from the fifth to the

eighth week. It shows that the tissues and blood are profoundly imbued with the poison, and betokens a fatal issue, very few cases recovering.

Vesicular syphilide occasionally constitutes a part of the general exanthem. When it occurs late in the disease it may correspond with the distribution of certain nerves, and then it is probably the result of syphilitic perineuritis. This form of vesicular syphilide differs from simple herpes in that there is more induration and staining, and that it is more persistent, and has a greater tendency to recur.

**Visceral syphilis.**—Very little is known of the visceral changes in the secondary stage of acquired syphilis. In the tertiary period they are among the best understood lesions. For the most part they are met with as chronic inflammatory thickenings, and lardaceous degeneration. The latter is more likely to develop when there has been long-continued suppuration, but it is not confined to these cases. The general cachexia is sufficient to account for its occurrence. The abdominal organs are the chief seat of the morbid process, especially the liver, kidneys, and spleen. As regards the essentially inflammatory exudations (*e.g.* in the liver) they are met with in four forms: (1) gummy tumours, (2) diffuse interstitial hyperplasia, (3) capsular indurations, (4) fibrous bands or seams, that divide the parenchyma into irregular nodular masses which simulate cancerous projections; but the latter are generally somewhat umbilicated in the centre, whereas syphilitic elevations are wanting in this feature. Syphilis is one cause of fibroid phthisis.

**Intracranial syphilis** includes (1) gummata which are generally found in the cortical portions of the brain; (2) chronic thickening of the meninges (pachymeningitis); (3) disease of the arteries (*q.v.*), which may lead to aneurism; or to thrombosis, and consequent cerebral softening. The symptoms to be

looked for are severe continued headache, some localised paralysis or spasm, and double optic neuritis. Syphilitic paralyses (*e.g.* of the cerebral nerves) are for the most part irregularly distributed and total.

**Intraspinal syphilis.**—Here also we find inflammatory induration, and occasionally gummata. Locomotor ataxy and lateral sclerosis in many cases owe their origin to syphilis. The peculiarity of these affections consists in their being to a great extent confined to certain columnar systems of the cord, in this way contrasting strongly with the generally irregular distribution of tertiary syphilitic lesions. The localisation seems to point to these so-called “scleroses” being dependent on degeneration of the nerve-fibres and fibroid substitution, rather than on ordinary inflammatory exudation. This view is further strengthened by the extremely chronic nature of the morbid process.

**Syphilis of the larynx.**—In the secondary stage we meet with an erythematous condition, mucous tubercles, and follicular ulcers. The inflammation subsides, or it passes into a more obstinate and destructive form, such as arises in the course of other tertiary manifestations. Tertiary ulceration of the larynx usually begins at the base of the epiglottis. It is accompanied by a good deal of swelling in the mucous and submucous tissues. The vocal cords and epiglottis may be entirely eaten away. The perichondrium suffers as well, it may be to such an extent that the cartilages necrose.

**Syphilitic eye affections.**—Of these, the one of most frequent occurrence is *iritis*, a rather late secondary lesion. Although the disease is centred in the iris it involves other structures, *e.g.* the choroid, and sometimes the retina. The effused lymph, which is in considerable quantity, is more lumpy, and darker in colour than in simple *iritis*. Permanent adhesions



to the lens capsule (posterior synechia) are liable to form, and by maintaining a constant state of tension predispose to recurrent attacks of inflammation. The natural hue and lustre are lost. The colour is a compound of the normal tint and that of the exuded reddish-yellow lymph; so that in a patient with dark eyes it becomes a rusty brown; and in one with blue it assumes a dirty-greenish appearance.

*Choroiditis syphilitica* is characterised by disseminated whitish patches, which at a later stage are surrounded by a zone of accumulated pigment. The pallor of these spots is due to absorption of the epithelial and stromal pigment, and to the vascular exudation. The re-deposit of the colouring matter follows the general law that guides inflammatory processes in highly pigmented tissues, *e.g.* the skin. It is not limited to syphilis, though in it it is unusually well marked. The subsequent changes consist of atrophy of the choroidal cells and vessels. The retina is more or less affected at the same time as the choroid, to which it becomes adherent.

*Retinitis syphilitica* presents itself as a diffuse exudation, which extends from the margin of the optic disc in an irregular manner along the course of retinal vessels. The outline of the cloudy whiteness is indistinct. This disposition contrasts strongly with the brilliant, highly refractive patches of albuminuric retinitis. Capillary hæmorrhages are not infrequent. The exudation is partly absorbed, and partly organised into imperfectly fibrillated tissue. Meanwhile, the proper elements of the retina waste; and the vessels get smaller and smaller, until only a few attenuated streaks can be seen traversing the optic disc, on their way to the fundus. When the atrophy has reached this degree the disc is small and pale. Scattered patches of choroiditis, as above described, may be seen in different parts of the fundus.



*Double-descending optic neuritis* is one of the symptoms of intracranial lesions, which are frequently of syphilitic origin.

**Congenital syphilis.**—The embryo may be syphilised from the first, through one or both parents suffering from the disease at the time of impregnation. Or the mother may become infected during pregnancy. In either case abortions are very common, but they are of greater frequency in the former. The placenta is often found to be extensively diseased, especially the fœtal portion. The chorionic villi are embedded in inflammatory exudations, which take the form of pale gummatous consolidations. This, together with the action of the virus upon the developing tissues, causes the death of the fœtus and consequent abortion, which is most common about the third month of pregnancy.

Should the fœtus survive until it attains viability, it may then be born alive or perish in utero.

In the latter case it generally shows unmistakable evidence of the disease. The skin is discoloured. The epidermis may be raised in blebs, or be undergoing desquamation subsequent to bursting of the bullæ of what was probably intra-uterine pemphigus. This must be distinguished from post-mortem maceration. When the child is born alive, it may die immediately after birth, or within the first few days of life. As a rule, however, nothing unusual is noticed until it is from three to eight weeks old. It is noteworthy that the secondary symptoms of congenital syphilis generally appear at about the same period after birth as those of the acquired disease after inoculation.

**Cutaneous eruptions.**—These tend to be moist. They usually consist of deep-red or copper-coloured blotches upon the palms and soles, and about the anus and genital organs. Scaly and tubercular syphilides

are not so well marked as in the acquired affection. At the same time, the rash is liable to be polymorphous. Pemphigus has been referred to.

**Mucous tubercles** (*vide* page 42) are very common. They are chiefly found near the verge of the anus, in the flexures of the groins, on the bucco-pharyngeal mucous membrane, and in the larynx.

**Affections of the mouth and nose.**—The mouth is subject to diffuse erythematous inflammation of the mucous membrane. It is also the seat of mucous tubercles, and superficial fissured ulcers which extend from the angles to the skin of the cheek. As these heal, they leave radiating pale cicatrices which may persist throughout life. The faulty development of the permanent teeth is the result of specific stomatitis.

The mucous membrane of the nose is thickened, and gives off a muco-purulent discharge. The perosteum and bones also suffer, hence imperfect growth of the nasal bones, which, together with defective expansion of the sphenoidal sinuses, explains the "flattening" of the bridge. (*Vide* page 282.)

**Affections of the eye.**—(1) *Acute iritis* is more frequent than is supposed. It is often not observed because not looked for. It is generally symmetrical, and occurs about the same time as the other early secondary symptoms, rarely beyond the sixth month. Since the anterior chamber is very shallow in infants, adhesions are liable to form between the lens capsule and the posterior surface of the cornea. There may be lasting evidence of this in the shape of a fine filament connecting the two structures, or of a central opacity on the front of the lens capsule (pyramidal cataract); but this is more common as a sequel of ophthalmia neonatorum than of congenital syphilitic iritis.

(2) *Interstitial keratitis* is a tertiary phenomenon. Most common about the age of puberty, it attacks both eyes, though there may be an interval of several months. Effusion takes place between the corneal laminae instead of upon the surface. It is first seen as hazy spots in the substance of the cornea, or it begins as a cloudy crescent, which rapidly becomes vascularised. As the spots enlarge, a more diffused or nebulous appearance is presented. Loops of capillaries form in the exudation in connection with the conjunctival and sclerotic vessels. As the inflammation subsides, the cornea clears up, first at the periphery, then more or less throughout; but, as a rule, there is a certain amount of haziness left, and, in severe cases, very considerable opacity.

Whilst the lymph is being absorbed or organised, the vessels shrink and finally disappear.

(3) *Kerato-iritis* is an inflammation of the cornea and iris. It is likewise a tertiary symptom, making its appearance from about the fifth to the eighth year.

(4) *Choroiditis* and *retinitis* are said to affect one or both eyes with about equal degrees of frequency. The pathology is the same as in acquired syphilis.

**Affections of the ear.**—Mucous tubercles are occasionally seen in the external auditory meatus, but they do not lead to any after-results. Congenital syphilis is the most common cause of permanent double deafness in children. This is due to chronic inflammation of the mucous membrane of the middle ear, and thickening of the membrana tympani.

The tympanum is blocked with granulation tissue, which contracts and overcaps the drum. Moreover, the ossicles become ankylosed, or even absorbed.

Otitis media may be accompanied by catarrh of the external meatus. This form of ear disease is liable to supervene on the subsidence of a kerato-iritis, but it also occurs alone. It seems probable that

in some cases of congenital syphilis there is disease of the labyrinth; *e.g.* cochleitis.

**Osseous lesions and teeth.** (*Vide* Diseases of bone, page 279.)

**Visceral lesions** consist of congestive and fibroid enlargement of the liver, spleen, etc., and gummata. Swelling of the spleen is very common. It may be temporary or permanent. The capsules of the liver and spleen are sometimes much thickened. It is said that perihepatitis from congenital syphilis is one cause of infantile jaundice and ascites.

"Abscesses" have been described in the lung and thymus gland. I have seen a gumma in the liver of a child twelve months old at the time of death. In the brain one meets with chronic inflammation of the cerebral arteries, meningitis, and hydrocephalus from "irritative dropsy" of the ventricles.

**General considerations.**—The secondary and tertiary symptoms of congenital syphilis are more frequently associated than in the acquired disease. Thus gummata may be met with at a very early age. Still, as a rule, there is a well-marked interval between the time of disappearance of the rash, mucous tubercles, snuffles, etc., and the outbreak of graver lesions, such as phagedænic ulcers, caries, and necrosis of bones.

Considering that congenital syphilis exerts its morbid action on young and growing tissues, it is not to be wondered at that 75 per cent. of the recorded cases of death from syphilis should happen in children under one year of age. How profoundly the whole system is steeped in the virus is shown by the rapid wasting of the body, and the cachectic look of the skin.

## CHAPTER XXII.

RICKETS—"SCURVY RICKETS"—"FETAL RICKETS."  
RICKETS.

RICKETS is a constitutional disease. Its chief manifestation is a lesion of bone tissue, occurring at a time of great developmental activity. It is looked upon by some as a symptom of *scrofula*, but it differs from it by the usual absence of other signs of that disease, such as suppuration and caseation, and by the whole osseous system being affected. In some of its features it resembles *congenital syphilis*, e.g. the fibroid induration of parenchymatous tissues, such as the liver, and the enlargement of the epiphyses of bones. In fact, inherited syphilis is considered a cause of rickets. The balance of opinion, however, is in favour of the two diseases being distinct.

**Natural ossification.**—To understand rightly the morbid changes occurring in the bones in rickets, we may briefly state what takes place in normal ossification at the epiphysis of a long bone.

In a vertical section there is seen: (1) A layer of hyaline cartilage; (2) a cartilaginous matrix impregnated with lime salts forming trabecular spaces, or alveoli which contain embryonic medulla and blood-vessels. This layer constitutes the *ossiform tissue* of Broca; (3) true bone.

The cartilage cells lodged in primary capsules enlarge and divide, and the new cells become surrounded by secondary capsules. A further segmentation occurs, and the secondary capsules undergo solution; broods of embryonic cells are thus formed, which quickly join adjacent groups by absorption of the matrix. The embryonic tissue becomes vascular



by blood-vessels shooting in from the canals in the true bone, which latter increases by the filling in of the alveoli.

These changes succeed one another so rapidly that the intermediate ossiform layer only attains a thickness of about  $1\frac{1}{2}$  mm.

The growing part is softer than cartilage, and hence gives way in "fracture through the epiphysis."

**Modified ossification in rickets.**—Let us now look at a similar section of a rickety bone. The layer between the cartilage and the bone is many times thicker than normal. The tissue of which it is composed has been termed *spongeoid* by Guerin, on account of its porosity and consistence. Unlike the healthy ossiform layer, it is irregular in outline, sending processes into the bone continuous with it. It is highly vascular, and sometimes contains islets of hyaline cartilage, partly explaining the isolated calcified patches seen along the line of the epiphyses. This spongeoid tissue at the surface joins with the subperiosteal *osteoid tissue* of Virchow, which is converted into a soft, thick, vascular substance that at a later stage becomes firmer and more adherent to the underlying bone. The central marrow loses a good deal of its fat.

**Microscopy.**—The primary cartilage capsules are unusually large, and the secondary capsules are very numerous and compressed; in fact, there is evidence of greatly increased activity in the initial process of ossification, whilst the later stages are not only delayed, but are for the time abortive.

Instead of the secondary capsules becoming dissolved, they are invaded by the calcifying process of the matrix, and thus the cells they contain are cut off from further active change. They become angular, are larger than bone corpuscles, and have no anastomatic canaliculi (Fig. 16).



No lamination is seen in the trabeculæ of the spongeoid tissue. The vascular channels of the old bone are continued into the cartilage, where they enlarge by absorption of the calcified tissue, and there, by joining together, form a system of intersecting canals filled with vascular embryonic marrow. In normal ossification the alveoli become occupied by fully developed bone; but in rickets the calcification, which is defective, occurs where it is not wanted, and



Fig. 16.—Zone of Proliferating Cartilage in Rachitis.

*a*, Cells pressed together and stained brown-violet with an aqueous solution of iodine, which stain is due to the glycogenic matter they contain; *b*, secondary capsule; section made in the fresh state and examined in water (*Corning and Ranvier*).

fails where it is. The corpuscles become angular, and the intercellular substance finely fibrillated. A similar condensation and fibrillation are noticed in the medulla of the cancellous bone, in the Haversian canals of the compact bone, and in the subperiosteal formation. The spaces everywhere containing this medulla enlarge by absorption of their walls, and thus, whilst the constructive process falls short, the destructive is

actively at work, and so the whole bone is rendered soft, and is readily bent by pressure.

In the osteoid tissue beneath the periosteum trabeculae form, and both in these and the osteoid basis the cells become stellate and anastomatic. Immediately surrounding the old bone in the more advanced stages are laminae of true bone, separated by a delicate connective tissue. This appearance is due to the increase and subsequent fibrillation of the medulla between the laminae which have been attenuated by absorption (Cornil and Ranvier).

When the morbid changes in the bones cease, ossification proceeds at a rapid rate, and at the epiphysis is completed before the usual time, accounting for the undue shortness of the limbs, since it is chiefly by the growth at the epiphyseal cartilages that the bones increase in length.

**Fracture.**—The callus thrown out in fractures during the rickety process consists of osteoid tissue, and does not pass through the intermediate stage of cartilage. It is large, and ossifies readily.

**Deformities.**—Even after all morbid action has ceased and ossification is completed, there is evidence more or less of the arrest of growth of the bones, which, coupled with the secondary deformities from pressure, make up the sum total of a rickety skeleton. It is true, as time goes on, a re-moulding of the bones takes place, so that curvatures partially or entirely disappear. Where they remain in the long bones, a buttress of compact bone is formed in the concavity of the curve, acting as a support, and so diminishing the liability to fracture. It is not uncommon for a long bone (*e.g.* the tibia, which is curved on account of its elongation from *diffuse* or *general* *ostitis*) to be mistaken for a rickety bone. A rickety tibia bends from inability to sustain the weight of the body, and the fibula follows its curve; whereas, in addition to

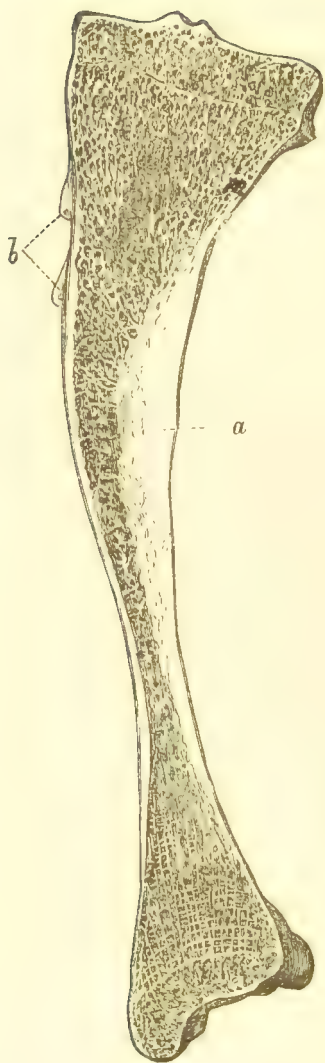


Fig. 17.—Tibia affected with Rickets.

*a*, Buttress of compact bone supporting the arch formed by the bending of the shaft. *b*, Small osteophytes. (One-third natural size.)



Fig. 18.—Tibia affected with Ostitis Deformans.

The scleros'd bone is thicker on the convex than on the concave side of the curve. The epiphyses are but little altered. The curve was single—antero-posterior. (One-third natural size.)

this cause, an elongated tibia becomes curved from the constant tension acting from the points of fixation at the ends of the fibula, the latter bone being healthy ; and this is not so much the result of actual bending, as re-absorption of bone in the concavity, and deposit along the convexity of the curve ; for, contrary to rickets, on vertical section we notice the compact tissue is much thicker in front than behind.

There are other points of difference which may be noticed here : the curve is single ; in rickets it is in two planes. The surface of the bone is rough ; in rickets smooth. The epiphyses are not so wide, perhaps, as the shaft ; in rickets they are much larger. The circumference of the shaft is more rounded ; in rickets flattened. The bone is longer than it should be ; in rickets shorter. (*Comp. Figs. 17 and 18.*)

*Fracture simulated.*—Sometimes in rickets, when the curvature is very marked in the leg, the shaft of the fibula is ossified to the tibia, simulating an old badly-set fracture of both bones. In the latter case, however, the curve is less general and uniform, and the other signs of a rickety bone are absent.

Although in rickets the whole skeleton is affected by the disease, the secondary deformities are more marked in some bones than in others, according to where the greater strain habitually falls ; and this, it is clear, will depend upon a variety of circumstances. If the child lie much upon the back, the occipital bone is flattened, and there is a corresponding prominence of the forehead ; for the bones of the cranium are easily displaced, since the delay in ossification protracts the time of closing of the fontanelles and the fixation of the sutures. Moreover, chronic hydrocephalus is by no means infrequently present, and this aggravates the cranial deformity. The pressure from without and within acting upon softened yielding bone, we see

why there is absorption here and there of the parietal and occipital bones (*craniotabes*).

In the spine there is usually antero-posterior and lateral curvature, with their resultant twisting. There will be a forward cervical curve and one large posterior dorso-lumbar if the child has been unable to walk, the body having been arched forwards from want of power of support; or the natural curves may be simply exaggerated. In some the lateral curve is in excess of the antero-posterior; occasionally it is so marked that one vertebra may eventually be half an inch deeper on one side than the other. A rickety spine with dorsal curvature can be told from the kyphosis of past caries by its more uniform curve, by the less complete (if any) consolidation of the vertebræ, and the smooth surface of their bodies.

It is obvious that this alteration in the shape of the spine must entail a displacement of the ribs and sternum, and so the general conformation of the chest. The sternum is thrown forwards and keeled, and the adjacent part of the rib cartilages prominent, the more so as there is some lateral depression arising from the sinking in of the cartilages nearer the ribs.

There is considerable thickening of the ribs at their junction with the cartilages, giving rise to the "beading," and these nodes lying in a curve on either side constitute the festooned "rickety rose-garland" (there is normally some beading of the ribs in their inner aspect). The slanting of the ribs must thrust down the liver (frequently enlarged), and so encroach on the abdominal cavity.

The pelvis is diminished at its inlet, having assumed a trefoil shape; the weight of the body displaces forwards the promontory of the sacrum and lumbar vertebræ, and the counter-pressure through the acetabula drives in those parts. A more irregular deformity, or lateral twisting, depends upon an



inequality of these forces. The femora show an exaggeration of the forward and outward curves; the forward, in particular, if the child has been carried much with the thighs across the arms of a nurse; the tibiæ are curved forwards, and either inwards or outwards; the fibulæ follow the tibiæ. In the upper extremity, we have in the same way to bear in mind the natural curves and the usual and unusual causes and direction of pressure.

The **joints** are frequently distorted, especially the knees, in the form of bandy-leg or knock-knee. This is the result of the alteration in the shape of the bones, and not of relaxation of the ligaments.

The **milk teeth** are cut late, owing to arrested growth, the dental enamel being very deficient. When cut, they crack, and decay earlier than natural.

**Visceral changes.**—The spleen, liver, and lymphatic glands may be found indurated, the two former at the same time being sometimes notably enlarged. The change is dependent upon a general increase in the interstitial tissue.

The enlargement has been thought to be due to lardaceous change; but it seems not to be identical, for fibrous tissue rarely, if ever, develops from the albuminoid substance found in the same organs in cases of protracted suppuration, cancer, syphilis, etc.; and, further, the new material in rickets is deficient in earthy salts; in lardaceous disease it is rich in them.

#### ACUTE RICKETS; SCURVY RICKETS.

Until a comparatively recent date the ætiology of "scurvy rickets" was little understood, hence the different names that have been provisionally employed in describing the more manifest signs; such *e.g.* are "hæmorrhagic periostitis" (T. Smith): "osteal or periosteal cachexia" (Gec). It may be stated that the bad hygienic conditions usually effective in giving rise to



rickets likewise suffice in certain cases to produce the morbid state under consideration, which may be defined as a cachexia or general malnutrition, having for its chief anatomical lesion an extravasation of blood beneath the periosteum.

The hæmorrhagic effusion and the frequent association with developed rickets justifies the employment of the name "scurvy rickets." The expression, "acute rickets," is less apt, since it implies that the pathological changes are necessarily modifications of those of ordinary rickets, and this is not warranted by facts.

Children suffering from the symptoms in question show a discoloration of the skin which has been variously described as sallow, yellow, muddy, and bluish-red; they are ill-nourished; the muscles are flabby and wasted. This is owing to a perversion of nutrition consequent on the particular constitutional state of the patient, and perhaps also owing to antecedent rickets (Barlow).

Though none of the bones are exempt, those of the lower limb are most commonly the seat of the effusion of blood which gives rise to a firm, elastic, painful swelling, liable to be mistaken for a deep-seated abscess. As a rule, "the thickening radiates from the junction area of shaft and epiphysis," and in this way differs from that of rickets, in which it is limited to the region of the epiphyseal cartilage. The entire limb or segment of a limb (*e.g.* the thigh or leg) may be enormously swollen, so copious may be the hæmorrhage. In the upper limb the swelling is often confined to the neighbourhood of the epiphysis; as before said, the bleeding takes place chiefly beneath the periosteum, so that the latter is raised from the bone. The entire shaft of a long bone has been known to be denuded in this way. Nor is the spontaneous rupture of blood-vessels confined to the periosteum; it is sometimes quite extensive in the soft tissues

around; and, what is more remarkable, it may entirely separate the shaft from one or both epiphyses, causing unnatural mobility in the part.

Dr. Barlow gives the following signs which serve for the differential diagnosis between scurvy rickets and ordinary rickets; *i.e.* so far as the bones are concerned. (1) The swelling is asymmetrical; if both sides are affected it is not to an equal extent; (2) the part is extremely tender; (3) there is loss of power in the limb, from (*a*) the pain; (*b*) separation of epiphyses when present; (*c*) mechanical interference from the extravasated blood. This on cursory examination may suggest the existence of infantile paralysis; (4) the swelling, even if very great, may subside rapidly. It is surprising how quickly the blood is absorbed, and the epiphyses again joined on to the shaft.

There is no effusion into the joints (Senator). Ecchymosis may be found in other parts, particularly the gums, and these either by the side of teeth already cut, or over others in process of being cut. There may be a purpuric eruption on the skin. (For a detailed description of the disease *vide* Trans. Med.-Chir. Soc., vol. lxvii. p. 159; 1883.)

#### SPORADIC CRETINISM; FŒTAL RICKETS.

The former name has been given to the diseased condition to be directly described, because it presents certain characters similar to those of endemic cretinism; *e.g.* the thyroid gland has been found abnormal in several cases, either ill-developed or goitrous, whilst in others it is absent. Then again the limbs are stunted in growth, and there is usually an excess (sometimes considerable) of subcutaneous fat, conditions which have been known to follow excision of the thyroid in young people, and which are believed to be allied to myxœdema.

The name "fœtal rickets" implies that the morbid state has some of the features observed in acquired rickets, such as defective bone formation and curvature of the limbs. There may be such a thing as true rickets in the fœtus, but if there is it is quite different from the disease commonly known as "fœtal rickets," which is now being discussed.

**Morbid anatomy.** —The osseous system is the chief seat of departure from normal growth and development. The diaphyses of the long bones are much shorter than natural, and they are often thick and curved. In some cases the epiphyses are enlarged, in others not. The ribs may be beaded at their junction with the cartilages, but this is not from the same cause which gives rise to the nodal swellings in true rickets; when present it is due to "an investing sheath of bone round the end of the costal cartilage" (Barlow).

There is an ingrowth of a process of fibrous tissue from the periosteum, between the shaft and epiphyseal cartilage. At the same time there is an arrested or abortive segmentation of the epiphyseal cartilage cells as shown by the absence of columns or rows of these cells. There is sometimes premature synostosis of the basioccipital and basisphenoid bones, in which event the base of the skull is short. The defective and perverted nutrition and growth observed in the cartilaginous moiety of the osseous system is not shared by the membranous, as the bones formed from membrane are well developed; "there is an overgrowth of membrane bone, with lack of metamorphosis of cartilage. It seems as though membrane and cartilage, instead of harmoniously working together to form bone, had broken alliance with each other, and pursued their own course to each other's detriment" (B. Sutton).

With regard to the thyroid, it may be quite normal, or it may be absent; or if present, larger or

smaller than natural; and since it is liable to such marked variations, it is clear that the morbid changes in other tissues are due less to a disturbance in its nutrition and function than to some more fundamental error probably common to both.

In sporadic cretinism, there is often an excessive quantity of fat in the subcutaneous tissues generally, but especially about the neck, where in addition to a general hyperplasia, there may be localised overgrowths.

Concerning the *etiology* of the disease very little can be said. It is only of comparatively recent date that the morbid anatomy has been studied with any degree of precision, and so far the results obtained throw very little light on the nature of the pathological process. The subject has been discussed in connection with rickets, not because it appears probable that there is any intrinsic relationship with that disorder of nutrition, but because it simulates it in some of its more manifest signs, *i.e.* in so far as the osseous system is concerned. Moreover, we have shown that the minute anatomy of rickets varies considerably from that of "foetal rickets," and the conditions of origin are very different in the two cases.

Again, further investigation is required to show whether or not there are grounds for belief in the hypothesis which supposes a generic affinity to exist between "foetal rickets" and cretinism. Although the disease is congenital, it does not follow that it is other than an accidental disturbance of the conditions which underlie normal development. Facts are wanting to prove that it is hereditary in the true sense of the term. Most of the cases are either still-born or they die soon after birth.

## CHAPTER XXIII.

## TUBERCULOSIS.

MANY different views have been held as to the structure and nature of tubercle. When first employed, it signified no more than that in certain diseased structures solid bodies could be seen with tolerably well-marked naked-eye appearances. No allusion was made to microscopical characters; in fact, the term was used in a loose generic sense, with a prefix to indicate its special origin; hence the expressions "scrofulous tubercle," "cancerous tubercle," and the like. But later it received a more restricted application, and came to be synonymous with the phrase "anatomical product of scrofulous inflammation."

The lungs of phthisical patients were, by the earlier pathologists, found post mortem to contain nodules varying in size, colour, and consistence. These they grouped under two heads: (A) minute, hard, semi-translucent bodies, from the size of a millet to a hemp-seed (*grey miliary tubercle*); (B) yellow caseous masses, the component elements of which had undergone fatty metamorphosis (*yellow tubercle*).

Virchow proposed that the term "tubercle" should be limited to the grey miliary bodies, wherever found, whether in the form of a widespread outbreak, or confined to the lungs as a part of the lesions of chronic phthisis; whilst others held it would be better to abolish the term altogether, or reserve it only for the cases of acute general tuberculosis.

Up to this time, patients, the subjects of chronic inflammations of a low type, inflammations which arose without apparent adequate local cause, and the

products of which showed a marked tendency to cascade and break down, were regarded as suffering from *scrofula* or *struma*; *i.e.* a general disorder which, although not explicitly ascribed to specific infection, was still considered as having features peculiar to itself.

The departure from the normal condition, concerning the causation and essential nature of which but little was clearly understood, received the equivocal appellation "scrofulous diathesis or dyscrasia." By some it was considered that the faulty state of nutrition was referable mainly or entirely to a depraved condition of the blood; by others to disordered metabolism of the tissues; and by a third section of pathologists to a morbid change common to both blood and tissues. The discovery of the bacillus tuberculosis by Koch, and the researches suggested by that discovery, have dispelled the uncertainty of our knowledge of the pathology of tuberculosis.

It is not now a question of the relation which scrofula bears to tubercle, since there is beyond doubt a definite explanation of the actual or exciting cause of so-called scrofulous inflammations. There can be no objection to the retention of the terms "scrofula" and "struma" so long as the anatomical lesions to which they refer are recognised as the result of the invasion of the tissues by the bacillus tuberculosis. The expressions "tubercular," "scrofulous," and "strumous" are therefore synonymous.

Although it is conceded that Koch's bacillus is the proximate cause of tubercular inflammation, the fact must not be lost sight of that some individuals are more prone to fall victims to the attack of the organism than others. The expression "tubercular diathesis" should be read as indicating simply a predisposition to such attack, or in other words, a low vitality or vulnerability, in consequence of which the tissues are unable to resist the successful invasion of



the bacillus, and evince but feeble efforts at repair. In many cases no doubt this predisposition is hereditary, though not in the sense of a specific transmission of disease as in syphilis, but as a perpetuation of an unstable condition of tissue just as in any other simple disorder of nutrition.

On the other hand, an individual may be born strong and of healthy parents, and yet through exposure to bad hygienic influences develop the predisposition. From the foregoing statements it follows that we should expect to meet, as is the case, with every degree of tuberculosis from slight affection of one tissue to the serious implication of many; and that in some cases the disease would terminate in recovery and repair, whilst in others it would pursue a continuous destructive course in spite of all that surgery can offer.

The products of tubercular inflammation have a marked tendency to undergo fatty degeneration and caseation. The caseous matter may remain inert for the rest of the patient's life; or after a varying period of quiescence, there may be renewed activity, causing further local destruction of tissue and entailing secondary infective inflammation in distant parts.

It is not uncommon to meet with old caseous masses, *e.g.* in phthisical lungs, surrounded by a halo of grey miliary tubercles. This disposition of morbid products may be accounted for by supposing that revived bacilli or their spores escape from the caseous centres and infect the tissues in the near vicinity. Should the organisms not find a suitable nidus for their development in the parts contiguous to the caseous deposit they may do so in more remote regions, *e.g.* acute tubercular meningitis may be the sequel of caries of the tarsal bones. It does not follow that all fresh outbreaks of tuberculosis are dependent on infection from old caseous nodules

even when the latter are present. It may be a repetition of the original infection, the bacilli entering the body from without. In the latter case they would be likely to attack by preference tissues whose vitality had been diminished by previous disease.

By their fermentative action tubercular bacilli generate in the fluids of the body, and in cultivation media, alkaloidal substances which collectively have been termed *tuberculin*. Tuberculin, although perfectly sterilised, has the remarkable property of exciting inflammation in tissues affected with tuberculosis. The process it sets up is inimical to the life of the bacilli, and it so far tends to eradicate the disease, but it cannot be relied upon in practice since experience shows that after apparent cure the mischief usually recurs. Moreover, its action on tubercular deposits is often so violent that the resulting inflammation causes necrosis of considerable tracts of tissue. There are two ways in which tuberculin may be supposed to exert a curative power over tuberculosis; (1) by the severity of the inflammation it induces, whereby both bacilli and tissue are destroyed; (2) by putting a stop to the functional activity of the bacilli, in the same manner that excess of alcohol affects the yeast plant in vinous fermentation.

*Tubercle compared with pyæmia.*—In some respects tubercle resembles pyæmia, especially in its mode of production, and more particularly in that form where it is artificially induced by exciting local inflammation. But the contrasts are many and important. Thus, in tubercle there is (*a*) the absence of embolic infarction and abscess; (*b*) the greater chronicity of its course; (*c*) comparatively little tendency to rapid softening of the disseminated products; (*d*) the undoubted fact of hereditary taint; or, in other words, the transmitted predisposition of the tissues to be affected by the pathogenic bacillus.

**Histology.**—At one time pathologists were content with describing tubercle as composed of a retiform tissue like that of a lymphatic gland; and the more so, as the grey granulations were mostly found where lymphoid tissue naturally exists, *e.g.* beneath the serous membranes and in the parenchyma of many organs, notably the lungs. Cornil and Ranvier assert that the fibrillar character of the meshwork enclosing the small embryonic cells is not natural, but is formed by the action of re-agents used for hardening sections, *e.g.* alcohol. The next step was the discovery of multinucleated and multipolar giant cells, the outrunners from which freely unite with one another and with the delicate fibres of the lymphoid tissue in which they are embedded. These giant cells cannot, however, be held by themselves to be characteristic, since they are not always present; and they have been found in other tissues, *e.g.* myeloid sarcoma, syphilitic gummata, and the granulations of simple ulcers. The giant cells of tubercle contrast with those of myeloid sarcoma in that they have numerous outrunners, and that their nuclei have a tendency to peripheral, or bipolar arrangement.

There can be no doubt but that the minute structure of tubercle is subject to variation. Thus it may be reticular, or not; it may contain giant cells, or not. A good deal depends upon the tissue affected, the stage of growth, and the extent of degeneration.

The general arrangement is this:—The giant cells, which occupy the centre of the tubercles, may be  $\frac{1}{60}$ th of an inch in diameter, and contain several nuclei; as many as thirty or forty it may be. Their processes unite into a fine net-work of themselves, and this is continuous with that of the adenoid tissue. The adenoid tissue varies in amount, constituting the

greater part of the tubercle, or forming only a narrow zone around the giant cells. The alveoli or spaces between the fibres are filled with small lymph cells (Fig. 19).

If Cornil and Ranvier are correct in stating that the fibrillar appearance of the matrix is artificially caused by the re-agents employed, then the continuity of the outrunners of the giant cells with the mesh-work of the surrounding tissue receives another explanation. Instead of uniting with the fibres of

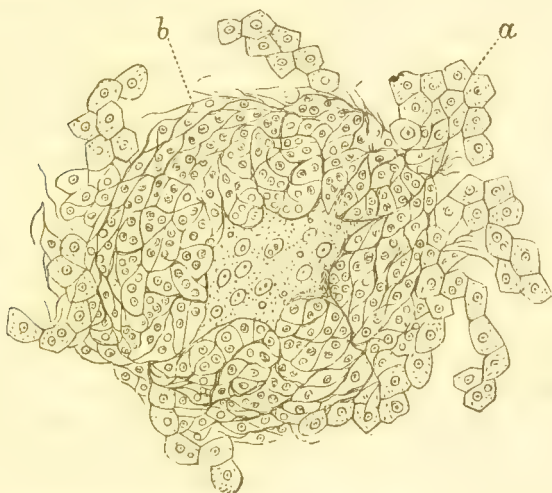


Fig. 19.—Grey Granulation of the Liver, from a case of Acute Miliary Tuberculosis.

In the centre of the tubercle is a multinucleated and multipolar giant cell. Its off-shoots join the stroma of the retiform adenoid tissue (*b*); *a*, liver cells.

retiform tissue these outrunners should be described as being simply embedded in inflammatory exudation. Our own observations lead us to believe that in most instances this interpretation should be accepted.

The entire tubercle is extravascular, or if vessels exist at all they become obliterated very early.

The centre soon becomes caseous (a constant

feature), and this depends (1) upon anæmia of the invaded tissue caused by the pressure of the overcrowding cells, and (2) upon an inherent low vitality.

**Origin of the giant cells.**—According to Klein, they start from the epithelial cells of the alveoli in the case of the lung, either by fusion of the protoplasm of contiguous cells, or by the continuous growth and arrested segmentation of individual cells. Another source is said to be the epithelioid lining of the lymphatics.

It may be remarked that caseation, formerly considered as characteristic of tubercle, is not confined to that morbid product, but occurs wherever the vitality of a tissue is slowly impaired. It is well marked in syphilitic gummata, and in tumours such as sarcoma and cancer. In these instances the elements undergo fatty atrophy from slow starvation.

Tubercular infection does not necessarily cause the development of grey miliary tubercles. The morbid product may consist for the most part, or entirely, of diffuse inflammatory infiltration in which new blood-vessels are formed.

**Bacillus tuberculosis.**—The organism is found in grey miliary tubercles, in diffuse inflammations and in the discharges from disintegrating tubercular growths, *e.g.* phthisical sputum. The bacilli of tuberculosis are not motile. They consist of straight or slightly curved rods about  $\frac{1}{3000}$  inch in length. They occur singly or in small groups. They are not crowded together like the bacilli of anthrax (Fig. 2).

“They form spores of an oval outline, both in artificial cultures and in the bodies of animals, and offer a high degree of resistance to drying, boiling, and the action of the gastric juice and of putrefaction” (Schenk). They can be cultivated in gelatine charged with blood serum. Animals inoculated with the cultures develop tuberculosis.



There are several ways of staining tubercle bacilli. For general use the Ziehl-Neelsen method is the best, both for discharges and sections. It consists of staining and counter-staining. The preparation is first stained with carbol-fuchsin. After being washed with water or alcohol, it is put in diluted sulphuric, nitric, or hydrochloric acid. When decolorised it is washed with alcohol and counter-stained with methylene blue. The bacilli are stained red and the groundwork blue.

**Tissues affected.**—The parts most prone to be affected are the skin, mucous membranes, bones, joints, testes, lymphatic glands, and the lungs.

In the skin we meet with (1) simple catarrh, or eczema; (2) limited superficial ulceration; (3) widespread ulceration. In some cases there is extensive undermining from destruction of the subcutaneous cellular tissue. The skin becomes purple and congested in parts from obliteration of the lumen of the vessels. This form is exceedingly obstinate and leaves thin broad cicatrices.

Catarrhal inflammation and ulceration of the mucous membrane of the *nose* is very common. It causes snuffling and offensive discharge (*ozæna*). It is most marked at the back of the nasal fossæ. Sometimes it extends to the soft palate, which may be fenestrated by ulceration. The bones may become carious or necrotic.

Children are very liable to catarrhal ophthalmia, and also to pustular conjunctivitis and phlyctenular corneitis. These affections are apt to recur again and again.

Scrofulous inflammation of the *bones* may only go so far as to cause a rarefying osteitis without supuration (*caries sicca*), but more often pus is freely formed. The bone is destroyed by the carious process, and it may be by necrosis as well. The cancellous



tissue is the favourite situation ; *e.g.* of the vertebrae, ends of the long bones, carpus and tarsus.

Tubercular disease of the *joints* commences either in the synovial membrane or in the bone.

In *strumous or tubercular orchitis* the epididymis is first affected ; both organs usually suffer sooner or later. The inflammation begins between the tubules

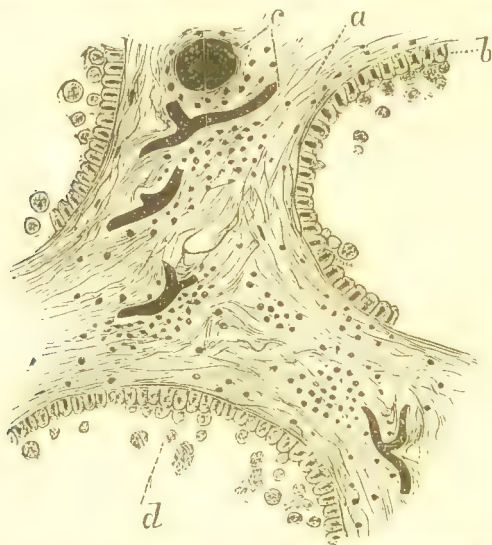


Fig. 20.—Chronic Interstitial Epididymitis (Scrofulous Testicle).

*a*, Intertubular tissue thickened and infiltrated with leucocytes ; the vessels, *c*, artificially injected ; *b*, epithelium lining tubules ; *d*, epithelium in process of disintegration.

(Fig. 20). Caseation and suppuration are common. The cord may be thickened. The vasa deferentia and the vesiculæ seminales and prostate may be enlarged.

Of the *lymphatic glands*, the submaxillary, cervical, mesenteric, and bronchial take the lead. The disease is rarely limited to one gland. It may lead to fibrous induration, but more likely to caseation and abscess. In the event of suppuration, the sores, which are slow to heal, leave indelible scars in the skin. In the neck

they cause great deformity. Scrofulous inflammation of the mesenteric glands is known as "tabes mesenterica."

The *lungs* may be thickly studded with miliary tubercles only, as a part of a general outbreak of tuberculosis. More often one meets with chronic phthisis in which there is interstitial deposit and catarrhal pneumonia; sometimes one predominating, sometimes the other. The degree of acuteness of the process and the stage of the disease explain the manifold appearances—grey miliary tubercles, caseating masses, fibroid tracts, and cavities, occurring in endless combination.

**Period of life.**—Scrofula is most common in childhood and youth, but no age is exempt. Those who have had scrofulous glands, or bones, or joints in their earlier years, often die of phthisis at a later period.

Sir J. Paget has described a *senile scrofula* of the bones, joints, and other parts, in which the pathological changes closely correspond to strumous disease of young subjects. It may be that such patients had an undeveloped predisposition to scrofulous inflammation in childhood, but that it only showed itself in old age, when senile decay was added to the original weakness of the tissues.

## CHAPTER XXIV.

## LUPUS.

THE results of Koch's experimental inoculation with tuberculin, of patients suffering from lupus, and the presence of granulations with giant cells, and of bacilli apparently identical with those of tubercle, force one to the conclusion that lupus is a tubercular affection. The comparative immunity from the disease enjoyed by old people, and the presence of the "apple-jelly-" like products are the two features in which it contrasts with the usual clinical and pathological history of tuberculosis. But this divergence, although not as yet satisfactorily explained, does not afford sufficient evidence for considering lupus as a disease *per se*. It is modified tuberculosis, and not a morbid process modified by tuberculosis.

It manifests itself in a peculiar form of chronic inflammation of the skin and mucous membrane. It is sometimes hereditary. It has a special proclivity for the first two decades of life. Its course and duration are alike indefinite. The face is its favourite seat, but it is by no means confined to that part. It also attacks the mucous membrane of the mouth, nose, and eyelids, usually by spreading from the skin. Several forms are described, but these are modifications of a common type, and not distinct varieties. If the disease go on to ulceration it is known as lupus exedens; if it stop short of this, lupus non exedens; if the ulceration be rapid and extensive, the word vorax is affixed, and so on.

It begins as a small red inflammatory nodule or thickening in the skin. This may disappear, and others form, or it may continue to enlarge. There

are usually several outlying tubercles, and as these increase in size they join one another and the central growth. The exudation is firm, and to the unaided eye looks like "apple-jelly," an appearance which is due to mucoid transformation of the inflammatory products. Some suppose that capillary thrombosis precedes the other changes, but it is difficult to say how far it is the cause or consequence of the inflammation. The microscope reveals a decided overgrowth of epithelium, both on the surface and in the glandular involutions; and an infiltration with leucocytes and homogeneous gelatinous material with giant cells scattered here and there. If a scraping from a lupus deposit be appropriately stained, bacilli are shown to be present.

The exudation on the surface dries up and forms an adherent scab, or it escapes as a purulent discharge; in the latter case the skin is as a rule ulcerated. As the inflammation subsides in the older parts, there is a tendency to cicatrise, to a greater extent than in rodent ulcer, but less than in tertiary syphilis.

But whilst the central part is healing, the peripheral may continue to spread, and in this way very extensive thin cicatrices may be formed. Whether there be ulceration or not, there is always loss of tissue, and, as a consequence, a certain amount of pitting or depression of the surface.

**Ulcerative lupus, lupus exedens.**—The tendency of lupous ulceration is to spread widely, rather than deeply; but there are exceptions to the rule. The enlarged papillæ are exposed, and then destroyed, and with them the glandular structures. In the more severe cases there is considerable discharge of pus, mixed with molecular débris. In these cases the edges are sharply defined, and the skin around is deeply congested, and the pain is often severe. Cicatrisation goes on slowly, for the inflammatory

new formation has little power of organising. That there is something peculiar in the nature of the morbid products is shown by the comparative readiness with which simple irritative exudation, caused by the means taken to destroy the lupus tissue, forms healthy granulations. The ulcers are mostly circular or sinuous in outline. There may be one or more. The cheek, the nose, and the eyelids are the parts most affected. After the wound made by caustics or scarification has completely healed, fresh tubercles are very likely to spring up in the neighbourhood of the scar.

When it attacks the face the ulceration is usually confined to the skin, subcutaneous tissue, and cartilages of the nose; but the bones may be extensively affected.

## CHAPTER XXV.

## TETANUS.

TETANUS is characterised clinically by tonic contractions of the muscles, commencing about the face and neck and spreading to the trunk and extremities, and pathologically by its bacillary origin.

**Ætiology and general pathology.**—In the majority of cases tetanus arises in connection with wounds, especially those of the limbs. The liability to the disease is in no way dependent on the severity of the injury; in fact, it is remarkable that in many instances the most acute onset and progress follow the slightest scratch. In hot climates it often arises without recognised injury.

Before the bacillary origin of the disease was established, it was held by some that traumatic tetanus was due to irritation of the nerves implicated in the wound, and that the spasms resulted from reflex irritation. In support of this view it was cited that now and then an attack was cut short by division or stretching of the nerves supposed to be at fault. The probable explanation of the phenomenon is that the injury inflicted on the nerve by the operation merely inhibited the spasm, much in the same way that nerve-stretching sometimes abolishes or lessens the pain and ataxy in tabes.

The clinical evidence that it is not simply a local disease is very strong.

(1) It is sometimes epidemic.

(2) The resemblance to strychnia poisoning, and still more to hydrophobia, is well marked.

(3) The spasms commence in muscles other than those in connection with the injured nerves.



(4) Some cases arise without apparent injury.

It is true the nerves have been found more or less locally congested and inflamed, but this must happen in all cases of wounds. I failed to find anything unusual in the median nerve in the case hereafter stated.

The bacillus of tetanus was discovered by Nicolaier after Carle and Rattone had discovered that the



Fig 21. Transverse Section of Spinal Cord (cervical region). from a case of very acute Tetanus. A portion of one of the anterior cornua of the grey matter, with the surrounding antero-lateral column, is seen.

*a*, Pia mater; *b*, anterior white column traversed by bundles of fibres going to form anterior root of spinal nerve; *c*, grey matter; *d*, blood-vessels; *e*, patches of exudation and softening.  $\times 85$ .

disease was communicable. Outside the body, earth mould is its chief habitat. It is anaerobic, and very slightly motile, it is nail shaped owing to its forming spores at one end. It is commonly found mixed with other organisms (symbiosis), but the proof of its

pathogenic identity is established: (1) by the fact that pure cultures can be obtained and the disease induced by inoculation with them, and (2) in that a temperature of  $80^{\circ}$  C. does not destroy its vitality, whereas it does that of its associates. To kill the spores it is necessary to expose them to steam at  $100^{\circ}$  C. for five minutes. The infective process set up by inoculation with the bacillus is said to be confined to the neighbourhood of the seat of injury.

**Condition of the spinal cord.**—Clifford Allbutt and others have described certain organic

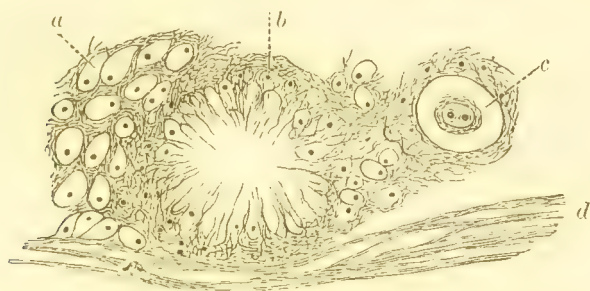


Fig. 22.—Portion of Fig. 16,  $\times 260$ .

*a*, Medullated fibres in cross section; *b*, focus of softening; the nerve fibres and neuroglia completely absorbed in the centre; *c*, exudation into perivascular lymphatic sheath; *d*, anterior cornual fibres.

changes, which in the main consisted of structureless exudation, and hyperplasia of the neuroglia. Cornil and Ranvier failed to detect anything abnormal. Of four cases examined by myself, two were to all appearance quite healthy; the third was simply congested; this might have been from asphyxial death; the fourth is represented by Fig. 21. The entire spinal cord, medulla oblongata, pons, and cerebellum (the cerebrum was not examined), were thickly strewn with patches easily visible to the naked eye. They were most numerous in the cervical part of the cord. They were readily stained with logwood and carmine.

Under the microscope they appeared as roundish homogeneous masses, distributed equally in the grey and white constituents of the centres. The nerve-fibres were in some places pushed aside by the exudation, in which, at the margin of the foci, delicate fringes of softened tissue were seen to be embedded (Fig. 22). There was moderate congestion. The perivascular lymphatic sheaths were in some places distended with a clear substance. There was singularly little cell-migration. The motor ganglion cells were healthy. The patient, a negro boy, *æt.* 11, under the care of Mr. Boon, of St. Kitts, had a gun-shot fracture of the *left* humerus and laceration of the median nerve. Tetanus appeared on the seventh day, and death occurred on the tenth. The spasms were chiefly confined to the *right* side of the body. Sections of the median nerve an inch above the seat of injury showed nothing abnormal.

## CHAPTER XXVI.

## UNION OF WOUNDS.

**Modes of union.**—1. By first intention or plastic adhesion. 2. By open granulation. (*Vide* page 30.) 3. By the union of opposed granulating surfaces. 4. By scabbing.

There is no such thing as immediate union, for however perfectly the edges of a wound are brought together, it is impossible for the open ends of the divided vessels to meet so accurately as then and there to re-establish the lumen. Besides this, the irritation caused by the injury sets up thrombosis in the mouths of the vessels, and the coagula must be absorbed before healing is complete.

1. **Healing by first intention.**—Take *e.g.* a simple incised wound in the skin and subcutaneous tissue. The edges of the wound gape on account of the elasticity of the skin. Bleeding takes place from the divided vessels until arrested by the formation of clots. It usually stops of itself, for exposure leads to spontaneous coagulation of the blood, and the vessels shrink from contraction of their muscular elements, and retract somewhat within their sheaths or bed of areolar tissue. On wiping the clot away from the wound, its small worm-like processes occupying the ends of the vessels are often drawn out, whereupon the bleeding recommences. It is immediately arrested, however, if the sides of the wound be brought together. There is now a thin layer of blood between the cut surfaces, and thrombi fill the vessels for a short distance beyond. Very soon a faint blush is observed at the margin of the wound, fading away into the healthy tissue. The visible redness, which is due to

paralysis of the vessels consequent on the injury, is an indication of a like condition in the deeper parts.

**Exudation stage.**—Leucocytes infiltrate the tissues and collect in the space between the divided surfaces, first occupying and then replacing the clot, which liquefies, and undergoes absorption. By this time there is slight tumefaction of the part, and the line of incision is covered with a film of fibrin, entangling blood corpuscles. In the meanwhile, the connective tissue becomes swollen, and its fibres less defined. As the inflammation subsides, the vessels regain their former calibre; numbers of leucocytes break up from fatty degeneration, and the débris is taken up by the capillaries, whilst others remain to take part in the process of cicatrization. As yet, the blood-vessels and lymphatics are blocked on each side of the column of leucocytes lying between the walls of the incision. The blood clot has disappeared, and the wound to all external appearance has healed, for at the end of two or three days there is well-marked cohesion between its sides. The firmness of the part is due to the condensation of plastic lymph (fibrin), that glues the inflammatory cells together.

**Vascularisation of the neoplasia.**—Loops and buds are given off from the vessels surrounding the wound, mainly in a direction at right angles to the latter. These approach from opposite sides, and, meeting midway, join by absorption of their contiguous walls. It is also possible that there is a free vascular new formation in the exudation, especially when there is a considerable tract of cells. In the meanwhile, the inflammatory corpuscles elongate. The wound is now united by vascularised lymph or *granulation tissue*, the capillaries of which are larger and more numerous than in the surrounding structures.

**Cicatrization.**—Some of the fusiform granulation cells remain as connective-tissue corpuscles.

Others fibrillate and contract, and contracting obliterate many of the vessels; nor does this cease until the scar is firmer and whiter than the normal tissue. Numbers of cells also disappear through atrophy from chronic starvation. If the adaptation of the edges of the wound has been accurate, and union unimpeded, the scar may entirely vanish in time.

**Failure of union** is brought about by any circumstance that adds to the necessary amount of traumatic irritation. 1. Poisoning of the wound, as in post-mortem cuts. 2. Mechanical obstacles to close apposition of the edges; (*a*) presence of foreign bodies or large blood-clots; (*b*) tension on the supports from muscular contraction, etc. 3. Bruising of the edges at the time the injury was inflicted, or subsequent irritation.

2. **Healing by granulation.**—There is no essential factor in this mode of healing that was not found in union by the first intention. The difference is one of degree and disposition rather than of kind. Taking the case of an incised wound, where the edges are allowed to gape, the following signs are observed.

Firstly, there is bleeding, which continues until the blood coagulates in the open vessels up to the next collateral branches. Then follow inflammatory hyperæmia and exudation. The transuded fluid is so rich in fibrin that it gelatinises on the surface, giving it a glazed appearance. But about the second day the exudation is too copious to be retained within the interstices of the tissues and on the surface of the wound; it therefore flows away as a pink serous fluid, the colour of which is due to suspended red blood-corpuscles and dissolved hæmoglobin. In a short time the discharge alters to a dirty yellowish-grey, in consequence of a greater proportion of pus cells, and the granular or flocculent débris of tissue elements that have died from the embarrassment of



the circulation in the superficial layer. It passes insensibly into genuine suppuration; but before the latter is quite established, the wound has undergone a decided alteration; small bright red elevated spots have made their appearance. These are vascular granulations, or groups of cells heaped up

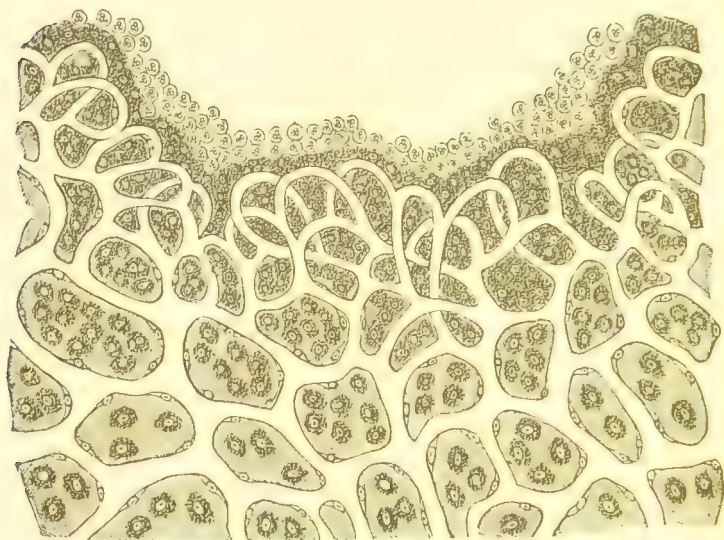


Fig. 23.—Diagram of Granulation of a Wound.

The layer of pus cells is represented as having been acted on by acetic acid, to distinguish the pus cells in the figure more accurately from the granulation cells (Billroth).

around festooned vascular loops given off by the underlying vessels (Fig. 23).

Suppose this layer of granulations to be folded up by bringing the opposite edges and surfaces together and the same arrangement is obtained as in healing by the first intention.

The granulations continue to grow until at length they reach the level of the surrounding surface, or pass beyond it. As the inflammation subsides, the discharge of pus diminishes, and organisation of the

granulation tissue takes place. Henceforth the case is one of a healing ulcer (page 30). The pus is derived from two sources: direct exudation from the vessels, and liquefaction of the uppermost stratum of the granulations. The drying-up of the secretion is not simply the result of removal of the cause of irritation. There is an inherent tendency to a typical end by organisation of the neoplasia, the same as obtains in normal development from embryonic tissue.

In **lacerated and contused wounds** portions of tissue are killed outright, for the vessels are twisted and bruised, besides being ruptured. Before union can take place, the dead parts must be thrown off; this is effected partly by the vital absorbent action of the granulation cells, partly by molecular disintegration of the sloughs (post-mortem decomposition).

Lacerated wounds inflame more than incised ones, for the injury to the tissues is greater, and the irritation is increased by the chemical products of the breaking-up of necrosed shreds when the entrance of germs has not been prevented; hence suppuration is more profuse and prolonged, and wider tracts are left to cicatrise. Although the cicatrisation is a conservative process, it may entail serious consequences from displacement of certain parts by traction. Ectropion, or eversion of the eyelid, is an instance of this.

Besides the extent of the injury, the nature of the tissue wounded has much to do with the rate of healing; as, for instance, in tendon, where the vessels are few, the anastomoses scanty, and the density of structure prevents a ready opening-up of collateral channels. In the last respect it resembles bone.

**3. Union of opposed granulating surfaces.**—This is much rarer than healing by the first intention or by open granulation. A *sinus* or blind fistula has only one opening on the surface. Its walls may be formed of healthy granulations, and yet union

may be indefinitely delayed from the tension of pent-up discharge, progressive disease at the bottom of the sinus, or the friction of the opposed surfaces; the last keeping up irritation and preventing the granulations from lying in contact in a state of rest. Instances of this are met with in sinuses beneath the scalp and pectoral muscles, and in the groin. Fixation of the muscles and external pressure are often sufficient to cause them to close.

The discharge from the granulations having been greatly diminished, the opposed surfaces are no longer kept apart. What little fluid lies between them is taken up by the vessels. Fibrinous exudation glues the granulations together, and henceforth the mode of union is that of healing by the first intention, except at the orifice, which is reduced to a small surface ulcer that cicatrises in the usual way.

4. **Healing by scabbing.**—The epidermis is grazed off, and the tips of the papillæ are bruised and slightly lacerated. There is very little bleeding. The coagulum which forms on the surface is increased by subsequent exudation. As the inflammation subsides, the scab dries up and breaks away at the margin; but before it is detached a new layer of epithelium covers the exposed papillæ. The wound is well before there is time for development of new vessels in the scanty effusion. In deep punctured wounds healing takes place by scabbing on the surface and by first intention beneath it.

We have yet to explain the mode of healing in wounds of certain tissues—tendon, muscle, nerve, cartilage, and bone.

**Wounds of tendons.**—The simplest case is subcutaneous division of a tendon, say that of the heel. The muscular end is drawn away so that a gap is formed; this is immediately filled up by sinking-in of the skin and effusion of blood.

The clot is quickly absorbed, and its place taken by plastic lymph, derived from the vessels of the tendon-sheath and adjacent areolar tissue. The tendon itself, on account of its density and defective vascular supply, takes but a minor share in the earlier reparative changes. The lymph, which is thickly set with leucocytes, not only joins the tendon end to end, but overlaps the edges, giving rise to a fusiform swelling. Vascular loops from the vessels of the surrounding tissue lie athwart the axis of the tendon, and, passing inwards join with those of the opposite side, and, later, with similar loops from the cut surfaces of tendon. In some respects this is similar to the growth of provisional and definitive callus in bone fractures.

The vascular cementing medium organises to connective tissue, and this undergoes cicatricial contraction. The scar-like tissue reverts pretty closely to the normal histological type, parallel bundles of fibres with interfascicular connective-tissue corpuscles.

*Lacerated wounds* are slow to heal, for they always cause death of a considerable portion of tissue, and it takes some time to throw off the softened shreds on account of the small supply of vessels in tendon. The superficial ulceration left after the deeper part is healed is troublesome, for the skin is bound down at the margin, and the contraction of the muscle keeps up irritation; hence the need of mechanical appliances to restrain movement.

**Wounds and injuries of muscle.**—Voluntary muscle is highly vascular, and its anastomoses are free and extensive.

When incised, muscle retracts more than any other tissue; the gap between the divided ends is wide. Bleeding, thrombosis to arrest it, and subsequent absorption of the clot, occur as in wounds of connective tissue. Granulation tissue is plentifully produced. The cells are mostly derived from the

blood-vessels, but it seems probable that the nuclei of the muscular fibres are aroused by the local irritation to renewed formative activity.

On division or rupture of the muscular fibres the contractile substance coils up somewhat within the sarcolemma, so that the ends become more or less club-shaped.

The ends of the fibres degenerate and split up longitudinally, and the nuclei to the same extent become granular from fatty metamorphosis. The degenerated products are absorbed, and the muscular fibres are seen to be lying in a bed of proliferating granulation tissue.

In the lower animals fusiform cells derived from the old muscle nuclei (Weber and Gussenbauer) and wandering corpuscles (Masłowsky) have been found to enlarge and become transversely striated, so that to some extent the union is by muscular fibre (Fig. 24). The same obtains in the human subject, although the repair is mainly effected by connective-tissue cicatrization.

If the divided ends of a muscle be approximated, a narrow seam of fibrous tissue is left. If there be much loss of substance, or the ends be allowed to separate, union is effected by a tendinous band, and the muscle remains more or less digastric.

When there is a considerable tract of divided muscle care should be taken to secure the opposed surfaces in close and accurate coaptation.

**Wounds and injuries of nerves.**—There are two remarkable facts connected with wounds and injuries of nerves. (1) The range of influence of nerve tissue over the mode of repair is very limited; the divided ends must be in close proximity to insure union by other than connective tissue. (2) Extensive destruction of nerve cells and fibres is compatible with continued function of the parts previously supplied by



them. A patient may be but little incapacitated by partial absorption and annular sclerosis of the spinal cord, the result of caries of the vertebræ (Charcot); and hæmorrhages, softening, and cysts of the optic thalami and corpora striata may leave very little

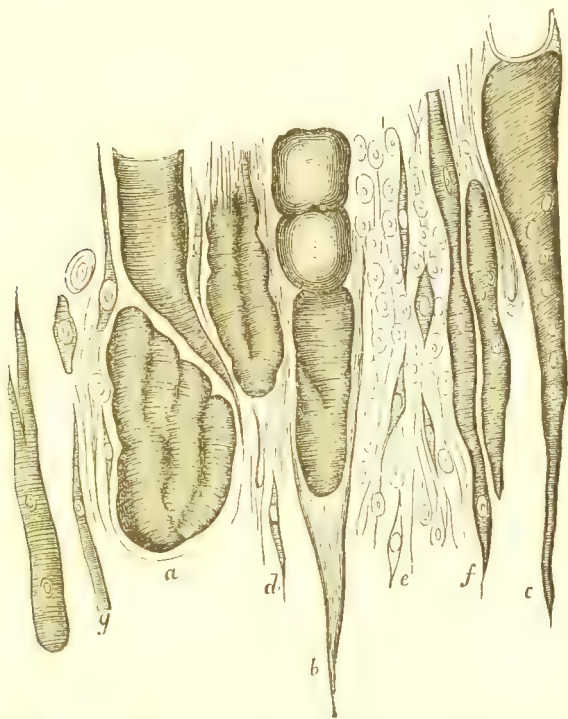


Fig. 24.—Ends of Divided Muscular Fibres, from the Biceps Musclev of a Rabbit, eight days after the injury.

*a, b, c*, Old muscular fibres; *a*, the contractile substance rolled up and balled together. The same way in the bundle above *d*: into the pointed cornet-shaped sarcolemma tube, *e*, there extends a series of young muscular nuclei, between which there is very delicate transversely-striated substance; *f*, young free muscle cells; *g*, two young ribbon-like muscular filaments; *h*, the same, of various sizes, isolated.  $\times 450$ . (From Billroth; after O. Weber.)

impairment of function. In these cases it seems more probable that the remaining elements take on increased action, than that new ones are formed.



As in muscle, the power of repair is greater in the lower animals than in man.

Brown-Séquard found that the paralysis caused by division of the spinal cord in pigeons disappeared after a time.

The approximated ends of a divided nerve may unite perfectly if disturbing influences be excluded.



Fig. 25.—Regeneration of Nerve. From the Frog, ten weeks after division. Development of young Nerve-cells from Spindle-cells.  $\times 300$ . (After Hjelt. From Billroth's "Surgical Pathology.")

This is seen after certain accidents and operations; *e.g.* the sensibility of the skin of the lower lip and chin may be restored after laceration of the mental nerve from fracture of the jaw, and the conductivity of the superficial cervical is re-established in the case of ligature of the carotid artery. Again, portions of transplanted skin become sensitive as nerve-fibres pass through the cicatrix to pick up or replace those in the graft.

Upon division of a nerve, degenerative changes ensue to a limited extent, less in the proximal than in the distal end, for it maintains its connection

with the central ganglion cells.

The white substance of Schwann breaks up, and the primitive sheath is applied to the axis-cylinder, the most durable part of a nerve-fibre.

Leucocytes infiltrate the part, and possibly the nerve nuclei increase in number. Some of the cells become fusiform, and, according to Schiff and Hjelt,

grow into nerve fibres with double contour, which pick up the ends of the old axis cylinders, and the nerve cicatrix is fully formed (Fig. 25). Short of this, the newly-formed fibres remain embedded in the connective-tissue scar, and whilst the distal end of the divided nerve undergoes progressive atrophy, the proximal end becomes bulbous, a reason for early operation in cases where nature has failed to effect the required union.

Dr. Reger, of Potsdam, sutured the ends of the musculo-spiral nerve four months after division, with complete success.\*

Billroth believes the proximal part of the axis-cylinder elongates and joins directly with the distal portion, the fusiform cells constructing the primitive sheath.

**Wounds or fractures of cartilage** unite by connective tissue, and, in the case of the costal cartilages, occasionally by bone. There is little, if any, reproduction of cartilage cells. (In ununited fractures of bone the ends of the fragments are sometimes capped with hyaline cartilage; this is in accordance with the fact that the enchondromata spring from bone or periosteum.) If a piece be split off an articular cartilage, the remaining portion proliferates at the seat of injury, the corpuscles divide and subdivide, and the capsules and matrix soften. The granulation layer thus formed organises to fibrous tissue. The loss of substance is never completely repaired, but a depressed cicatrix remains. When the injury implicates the adjacent synovial membrane vascular loops grow into the granulation tissue, but they disappear as cicatrisation advances.

\* *Lancet*, May 31, 1884, p. 994.

## CHAPTER XXVII.

## FRACTURES OF BONE AND PSEUDARTHIROSIS.

**Simple fracture.**—When the shaft of a long bone is broken, there is *bleeding* from the vessels in the central medulla, Haversian canals, and periosteum, and also from the soft tissues round about, if these be lacerated by the displacement of the fragments. If the bleeding has been excessive, fluctuation, or bogginess, may be felt over the seat of fracture, and there is ecchymosis of the skin, and perhaps also bullæ filled with blood-stained serum. The staining of the skin becomes more marked after a while, for the blood corpuscles break up, and the colouring matter is diffused through the tissues.

When the bone is deeply placed, or the break in it is a mere fissure, there may be no discoloration of the skin, or, at the most, only a light-yellow tinting from hæmoglobin that has passed through the usual changes before it has reached the surface. Such may be seen in Scarpa's triangle a few days after intracapsular fracture of the neck of the femur.

Possibly a portion of the coagulum around the ends of the fragments organises, but certainly the greater part is re-absorbed.

*Inflammatory changes.*—The swelling of the part is increased by exudation, which often causes œdema of the soft structures. There may be redness of the skin from hyperæmia. The inflammatory changes set in immediately after the injury, and the products at first mingle with the extravasated blood, which, together with the serous portion of the exudation, is removed by absorption. Then it is that the more plastic fibrinous material can be felt as a firm substance embedding the broken ends of the bones. This

will be about the end of the first week, when it is said the callus begins to form.

It must not be supposed that up to the present there has been a simple sequence of three events:— hæmorrhage and absorption of blood, serous effusion and absorption, and the beginning of callus-formation. These processes overlap one another, as it were, and it is only the maximum development of each that is noted as a distinct phase.

The result of the inflammation is that the medulla (subperiosteal, central, and interstitial or Haversian) has been converted into embryonic tissue, the cells of which lie fixed in a bed of firm intercellular substance.

The early induration, which is more marked than in any other situation, takes place under the physiological law that rules the natural growth of bone.

The cells of the callus are derived from the blood-vessels, the bone corpuscles probably taking no active part in the process.

So far as the bone is concerned, the embryonic or granulation tissue is limited externally by the periosteum. It forms a thin layer between the ends of the fragments, and fills up for a short distance the

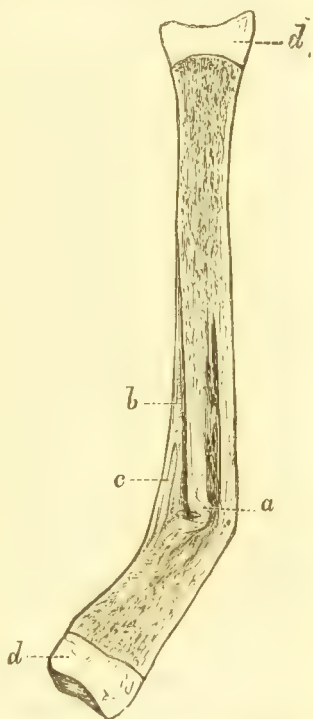


Fig. 26.—Simple Greenstick Fracture of Radius. About three weeks after the injury.

*a*, Compact bone of shaft; *b*, callus; *c*, cartilage formed in the callus; *d*, cartilaginous epiphyses; the callus is chiefly deposited in the concavity of the fracture-curve. (Natural size.) St. Mary's Hospital Museum.

open ends of the Haversian canals. It also blocks the medullary canal. The fat cells of the central medulla are broken up and their contents absorbed.

The inflammatory new formation is in greatest amount opposite the fracture, *i.e.* where the irritation from the injury is most intense; from this point it subsides gradually, so that, if the broken ends be in accurate coaptation, they will be ensheathed by a regular fusiform collar or ferrule.

**Cartilaginous transformation of the callus.**—Some bones are developed directly from embryonic tissue, and all grow in thickness from the same; but many, *e.g.* the long bones, ossify from a cartilaginous basis until their full length is attained. So, in fractures, the callus formed of indurated granulation tissue may ossify forthwith, or pass through the intermediate state of cartilage (Fig. 26).

According to Cornil and Ranvier, the callus of simple fractures is converted into cartilage; that of compound fractures is not.

The irritation in a compound fracture, where there is more or less suppuration, may be too great to allow of the necessary conditions (of which one is rest) for the transformation of granulation tissue into cartilage.

Age is likely to influence the event; for in adults the natural formative activity of cartilage is almost *nil*, and cartilage tumours do not often show themselves late in life. On the other hand, cartilage is always present in the growth of osteophytes about the joints in chronic rheumatic arthritis.

Cartilage first appears at the upper and lower borders of the callus, both in the periosteal and central portions. It continues to grow until the chondrification is complete.

**Vascularisation of the callus.**—The blood-vessels of the bone and periosteum give off loops into

the young peripheral callus ; those of the bone only into the central callus.

The new vessels lie at right angles to the axis of the bone in each case.

Vascularisation of the callus between the opposed fragments is much slower than on the surfaces ; for the old vessels are pent up in the Haversian canals (except at the torn ends, which are plugged with clots), the rigid walls of which retard dilatation and lateral looping ; and hence it is some time before the continuity of the vessels in the two fragments is re-established.

**Ossification of the callus.** -Whether cartilage be formed or not, lime salts are deposited around the blood-vessels, embedding the immediate granulation cells (osteoplasts). This follows in close order upon the growth of the vessels, and, in the superficial callus, advances from the bone and periosteum at the same time. The primary trabeculae of bone form moulds around the vessels.

As ossification advances, the osteal and periosteal spiculæ meet and widen, but the ossified callus never attains the density of compact bone ; for, before that stage is reached, retrogressive or absorptive changes set in.

The osseous columns in the central and external callus lie, like the vessels, parallel to the transverse axis of the shaft of the bone.

By the end of the first week, or a little later, ossification has commenced ; by the end of the third it is sufficiently extensive to enable the callus to keep the fragments in apposition, but an extraneous force, *e.g.* the weight of the limb in fracture of the femur, might bend or break the callus.

If the callus be changed to cartilage, ossification follows the physiological type. The primary capsules contain secondary capsules, which, dissolving, set free



broods of embryonic cells derived from proliferation of the cartilage cells. By absorption of the capsules and matrix, festooned passages filled with indifferent cells are opened up. Into these blood-vessels grow, whilst lime salts are deposited in the matrix. So far there is only calcification. True ossification is established by the enclosure of osteoplasts as bone corpuscles in the ossifying matrix, corpuscles which unite by their processes, and lie in lacunar spaces that join one another and the central canals of the new Haversian systems.

**Provisional and definitive callus.**—Dupuytren gave the name *provisional* to the callus formed about the ends of the fragments (that which causes the fusiform swelling on the surface, and blocks the medullary canal), since it provided for the union and support of the fracture pending the construction of the final or definitive callus between the broken surfaces.

The amount of provisional callus depends chiefly upon the extent of the injury to the bone and soft parts. In comminuted simple fracture it is very large, and welds the fragments into a continuous mass. In children it is more largely developed than in adults.

If there be no displacement of the fragments, which require but little support, as in fissured fracture of the skull, it may be so small as to escape detection. The provisional callus so far disappears that the central canal is re-established and all external signs of the fracture are lost, providing there has been little or no displacement of the fragments. Its complete absorption requires months.

*Definitive callus*, though slow in forming, far exceeds the provisional in density; indeed, it becomes more compact than the old bone, hence the saying, "A bone does not break twice in the same place."

As the result of the injury, rarefying osteitis (pages 257-269) is set up in the ends of the fragments. This goes on until the bone is quite porous, and the canals, enlarged by absorption, are filled with vascular granulation tissue. The blood-vessels from the opposite fragments meet and unite. Lime salts are deposited around them, so that the new bony trabeculae, unlike those of provisional callus, lie in the long axis of the bone; and the continuity of the Haversian canals is once more established.

The rarefying or absorptive osteitis subsides into a sclerosing or constructive osteitis, and when this ceases repair is complete. The provisional callus is absorbed *puri passu* with the formation of the definitive.

The amount of provisional callus formed, and the extent of the rarefying osteitis, vary as the intensity of the inflammation, and this as the degree of injury.

**Changes in the periosteum, etc.**—The medullary layer is converted into granulation tissue, and thence to callus.

The fibrous layer becomes indefinite in outline, and commonly lost in the softened mass. In the latter event, a new periosteum is formed from the superficial portion of the callus.

The development of new bony callus is not confined to the periosteum and bone; the tendons and connective tissue of the muscles are likewise osteoplastic. In simple fracture without displacement they do not enter into the process of repair, but where there is much laceration of the soft structures they are very active. The muscular fibres undergo absorption corresponding in extent to the osteoplastic change in the interstitial tissue.

When a fracture passes through a strong tendinous insertion, the callus at that part is increased by the physiological tension upon the tendon; and when ossified, it may remain as a permanent



Fig. 27.—Defective Growth of Radius, the result of Fracture of the Lower Epiphysis.

A ridge of bone is seen at the seat of union. (Reduced one-half.)

osteophyte, whilst in the other parts it is completely absorbed.

**Fracture through epiphyseal cartilage** is in reality fracture at the line of junction of the cartilage with the bone, so that one fragment carries the whole or greater part of the cartilage with it. This is the reason why epiphyseal fractures, so common in children, do not, as a rule, lead to a stunted growth of the bone. Severe injury may cause the cartilage to be entirely converted into embryonic tissue, and this again into ossified callus, so that at one end the bone is arrested in its growth lengthwise, but as a rule, a part of the cartilage escapes the inflammatory change.

The cartilage developed in the callus probably ossifies throughout. It does not appear to possess the physiological property of continuous segmentation and reproduction (Fig. 27).

In fracture through the epiphysis of the lower end of the humerus, the provisional callus partly fills the coronoid and olecranon fossæ, and for a time checks the movements at the joint. The crepitus is not so hard and grating as in fracture through bone.

**Failure of ossific union.**—The causes of non-union are constitutional and local. The *constitutional causes* are those that impair the healthy nutritive and formative activity of the tissues in general, and in one disease (mollities ossium) bone tissue in particular. Chronic Bright's disease, tertiary syphilis, and cancerous cachexia may be cited as likely to prevent bony union, and permanently so. Fractures are slow to unite during acute specific fevers, and afterwards if convalescence be protracted.

The *local causes* are: (1) Those that prevent coaptation of the fragments; (2) movement of the fragments one upon the other; (3) defective vascular supply.

1. **Separation of the fragments** may be effected by (*a*) muscular action, as in fracture of the patella and olecranon; (*b*) the hydrostatic pressure of the effusion into the neighbouring joint, *e.g.* the knee; (*c*) the interposition of a foreign body, such as muscle, tendon, a detached portion of bone, a tooth in fracture of the jaw, or the necrosed end of one of the fragments. It is said that in fracture of the patella the superposed bursa dips in between the broken surfaces.

Wide separation by muscular action means that the new formation connecting the fragments is, at the part most distant from the bone, removed from the sphere of influence of the physiological stimulus to ossification possessed by the bone and periosteum.

2. **Movement of the fragments**, the principal cause of non-union, entails more than the absence of coaptation. In a case of transverse fracture of the patella where the patient died from cellulitis of the limb following the use of Malgaigne's hooks, I found the fractured surfaces thickly studded with needle-like processes of new bone, a sixth of an inch in length. Now, movement would tend to break off the tips of these minute stalactites, but would not prevent

their fusion into a continuous layer by lateral growth.

It seems strange at first sight that movement should cause an exuberance of callus, and yet in some cases prevent osseous union. The explanation is probably in the degree and continuance of movement. If the callus suffices to lock the fragments together, it will ossify; if it fails to do this, it will not.

3. **Defective vascular supply.**—I believe this has little to do with the failure of union in the case

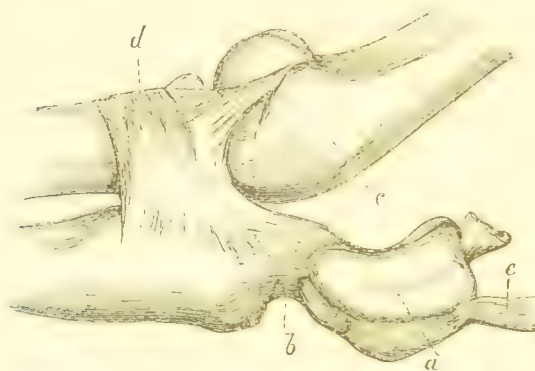


Fig. 28.—Ununited Fracture of Olecranon.

*a*, Upper fragment; *b*, ligamentous band joining the two fragments; *c*, humerus; *d*, olecranon ligament; *e*, triceps tendon.

of the patella and olecranon. In fracture through the anatomical neck of the humerus, the blood supply can only come from one surface; and in intracapsular fracture of the neck of the femur, the cervical ligament (periosteum) is usually torn, and the head of the bone gets very little nutriment through the ligamentum teres, even if the latter is not ruptured.

### **Pseudarthroses, or false joints. Causes.**

(1) Ununited fracture; (2) unreduced dislocation.

There are two varieties of false joint as the result of ununited fracture: (*a*) ligamentous, (*b*) diarthrodial.

*a. Ligamentous pseudarthrosis* is seen in fractures of the patella, olecranon (Fig. 28), acromion, and neck of the femur within the capsule.

Unless the fragments be separated by serous effusion into the neighbouring joint, *e.g.* in the knee, in many cases of fracture of the patella, the interval between them is soon filled up with soft granulation tissue, the vessels of which are derived from the broken surfaces and the surrounding structures. As the granulation tissue organises, the fibrous bundles lie parallel to the line of greatest tension, *i.e.* from fragment to fragment.

The distensibility of the young connective tissue accounts for the separation of the fragments subsequent to their having been in pretty close apposition.

Cartilage cells may sometimes be found in the meshes of the fibrous tissue at the fractured surfaces.

*b. Diarthrodial pseudarthrosis.*—The movement of one fragment upon the other may be so free as to prevent the growth of granulation tissue between the surfaces, or, if it has formed, to cause its re-absorption. This variety of false joint is found in the long bones. The medullary cavity on each side of the fracture is filled with callus, which may change to cartilage. The internal callus ossifies, but instead of being re-absorbed, as is usually the case when bony union occurs, it remains as a permanent compact mass. This result is due to the friction kept up between the fragments, inducing condensing osteitis. Meanwhile the granulation tissue formed from the soft parts around the fracture organises, and the broken ends are enclosed within a dense fibrous capsule. When the cartilaginous callus within the medullary canal ossifies, an irregular layer of hyaline cartilage is sometimes left upon the surface (Fig. 29). A natural joint is further simulated by the change in shape of the fragments as they rub together, the



cup-shaped hollow of one receiving the rounded extremity of the other. If the movement is mainly in one plane the joint is ginglymoid. The cavity of the false joint contains a little serum (not synovia) exuded from the vessels of the capsule.

### **Pseudarthrosis from pathological dislocation.**

—The cartilage of the old joint is wholly or in great part removed before the dislocation occurs. In *caries of the hip joint* the head of the femur is more or less absorbed, as well as the rim of the acetabulum, on the side of the dislocation where there are softening from caries, and atrophy from the *continued pressure* of the head of the femur; hence the luxation usually takes place gradually. The capsule of the hip joint, softened by inflammation, is readily stretched by the localised tension upon it; and at last it ruptures or is entirely destroyed by the disease at this part.

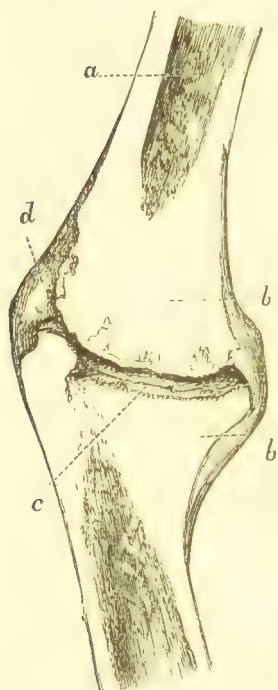


Fig. 29.—Ununited Fracture of Humerus. Diarthrodial False Joint.

*a*, Medullary cavity of the bone; *b*, compact bone closing the medullary cavity next "the joint"; *c*, layer of cartilage; *d*, fibrous capsule containing a nodule of bone.

Subsequent to the dislocation the caries may subside, and the granulation tissue become fibrous, and form a new capsule around the displaced bone.

Then the cotyloid cavity becomes shallow, from atrophy of its margin and filling in of the hollow with connective tissue or bone. The hip bone is rough from osteophytes for some distance beyond the primary

seat of disease. Where there is greatest movement (*intermittent pressure*) of the femur upon the hip, the irritation causes a buttress of bone to be thrown out, that acts as a support to the dislocated femur. This constructive process may go on whilst the caries of the bones round about is progressing (Fig. 46). In *Charcot's disease* two factors may be concerned in the dislocation: (1) Erosion of the bones; (2) stretching of the capsule from serous effusion into the joint.

**Pseudarthrosis from traumatic dislocation.**—Here the dislocated bones are healthy at the time of the accident. The subsequent changes are: (1) Construction of a fibrous capsule about the false joint; (2) alteration in the shape of the bones from mutual pressure; (3) partial or complete absorption of the articular cartilages; (4) diminution in depth of the old articular depressions (glenoid, cotyloid, etc.); (5) formation of a buttress of support for the dislocated bone.

#### UNION OF COMPOUND FRACTURES.

A compound fracture may be converted into a simple one by immediate closure of the wound in the skin or mucous membrane.

The laceration of the soft structures, which is considerable, gives rise to a good deal of extravasation. It may possibly cause suppuration about the fracture after the external opening has closed.

Besides the extent of the injury to the soft tissues, splinters of bone may be detached, and the ends of the fragments may necrose from stripping off of the periosteum, or consecutive inflammation. Taking an uncomplicated case of compound fracture, with suppuration, the bone, periosteum, and soft tissues implicated are acutely inflamed. The purulent exudation is at first mixed with disintegrating blood-clot. It bathes the broken ends of the bones, which are bare at the

bottom of the wound. As the inflammation subsides a layer of vascular granulation tissue lines the whole interior of the wound. This for a time continues to furnish pus, but at length the granulations from opposite sides come in contact and their vessels join.

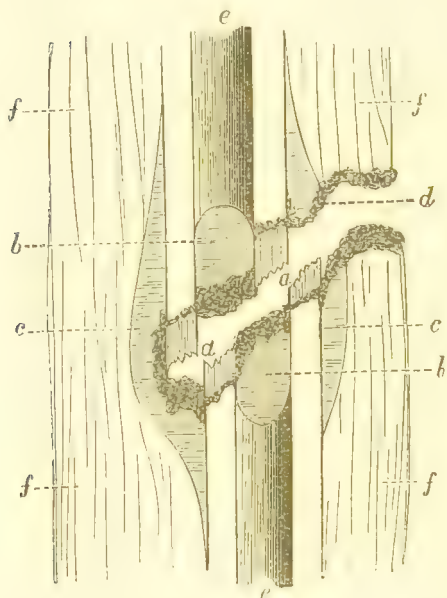


Fig. 30.—Diagram of Fracture of a Long Bone, with external Wound; longitudinal section. (Natural size.)

*c, c,* Bone; *f, f, f, f,* soft parts of the limb; *a, a,* necrosed ends of the bone; the darkly-shaded part represents the granulations which line *d*, the wound that opens cut-wardly, and secrete pus; *b, b,* internal callus in the two dislocated ends of the bone; *e, e,* external callus. (After Billroth.)

The inflammatory new formation passes through the usual changes. The cells that remain are embedded in firm inter-cellular substance, in fact, the whole is converted into a mass of callus. The callus organises into bone without the intermediate formation of cartilage (Cornil and Ranvier). It rarely ends in fibrous union of the fracture. The bone, periosteum, and fibrous structures in the vicinity are all active in the osteoplastic process, so that the amount of provisional callus is very great.

The rarefying ostitis of the ends of the fragments is more extensive than in simple fracture, and this delays the time of completion of the definitive callus.

**Compound fracture, with necrosis.**—Portions of bone may be cut off at once from all vascular supply, and lie loose at the seat of fracture; or necrosis

of the ends of the fragments may ensue from the violence of the injury, stripping off the periosteum and causing extravasation into the Haversian canals, or from arrest of the circulation, consequent on acute inflammation (Fig. 30).

Splinters of bone may retain their connection with the periosteum, and, surviving the effects of the injury, help in the process of repair. Even if completely detached they should be fixed in position, for in aseptic wounds such fragments may live and re-unite. This is well exemplified in the operation of trephining, where the button of bone, after being kept in warm boracic solution, is replaced. Necrosed portions of bone, unless removed, keep up irritation, and whilst they stimulate to increased bone formation

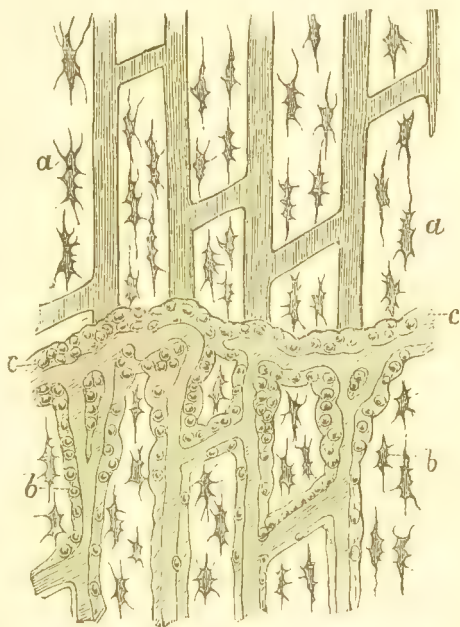


Fig. 31.—Diagram of Detachment of a Necrosed Portion of a Bone. Magnified 300 diameters. *a*, Necrosed portion of bone; *b*, living bone; *c*, new formation in the Haversian canals by which the bone is detached. (After Billroth.)

ation they prevent closure of the sinuses. If the ends of the fragments lose their vitality, union of the fracture is greatly delayed, but it may take place sufficiently to allow of restoration of the function of the bone whilst the sequestra are retained; *i.e.* the mass of new bone thrown out from the outer surface of the living portions of the fragments and the soft tissues around,

may bridge over the gap that holds the sequestra, so extensively as to leave but one or more narrow apertures (cloaca) for the escape of the purulent discharge from the granulations within.

So long as the sequestra remain imprisoned suppuration will continue; but if sequestrotomy be performed, even after the lapse of years, the sinuses will close and the case end favourably. Nature is unequal to the liberation of the dead pieces, for before they are detached a mantle of new bone has been deposited around them, and the sinuses have contracted too much to allow of their extrusion by the pressure of the granulations. The casing of new bone becomes very thick and dense, and the cavities left after removal of the sequestra fill up slowly on account of the dearth of vessels. The sequestra are quite characteristic. They present at one end a brittle fracture, at the other a worm-eaten appearance.

**Spontaneous fracture.**—By this we mean that the fracture results from an injury wholly inadequate to the breaking of a healthy bone. The force is almost always applied indirectly. The fracture sometimes occurs without the knowledge of the patient.

*Causes.*—(A) More or less general. 1. Senile osteoporosis. Here the bones are very brittle, the compact tissue is wasted, and the spaces of the cancellous tissue are large and filled with fat. 2. Mollities ossium. In this disease there are usually multiple fractures. 3. Rickets. 4. Charcot's disease, and other nervous affections. The readiness with which the bones often break in lunatics is well known. 5. Hereditary fragility.

(B) Local causes. 1. Absorption of the bone by a new growth, or the pressure of an aneurism. 2. Congenital syphilitic dystrophia, causing separation of the epiphyses. 3. Syphilitic gummata. 4. Fatty atrophy from disuse, *e.g.* in long-standing joint

affections. 5. Alteration in the angle that the neck of the femur makes with the shaft in old people. This places the bone at a mechanical disadvantage when force is applied to the long arm of the lever. (The fulcrum is at the hip joint, and the resistance at the seat of fracture.) 6. Separation of the epiphyses from hæmorrhage in "scurvy rickets" (pages 180-191).



## CHAPTER XXVIII.

## INJURIES AND DISEASES OF THE SCALP.

Circoid aneurism, nevus, atheromatous cysts, and subaponeurotic cellulitis are described elsewhere. The parasitic diseases are beyond the scope of this work.

**Hæmatoma.** —Hæmatoma is an extravasation of blood sufficient in quantity to give rise to a boggy or fluctuating swelling. There are two forms of cephalhæmatoma, subpericranial and subaponeurotic. In the former the effusion is beneath the periosteum; in the latter, beneath the tendinous expansion of the occipito-frontalis muscle. Subpericranial hæmatoma is limited to the bone over which it commences, for the periosteum is too firmly fixed at the sutures to allow of its further separation. Subaponeurotic hæmatoma, whilst usually confined to a circumscribed area, is occasionally diffused over the cranial vertex from the superior curved line behind to the brow and root of the nose in front. It is impossible to tell by manipulation whether a localised hæmatoma is beneath the pericranium or aponeurosis. Necrosis is more likely to follow the former, but it is not common in either case, unless the bone is severely injured, for the vascular supply is chiefly derived from the meningeal arteries. The extravasated blood passes through the usual process of disintegration prior to absorption.

But while there is yet fluctuation, inflammatory lymph is deposited at the base and margin of the swelling, so that the latter is surrounded by a vascular granulation membrane. The exudation from this mingles with the liquefying clot, and it may end in suppuration, but more frequently it is reabsorbed. The inflammatory induration terminates abruptly on

the side of the hæmatoma, whereas it gradually subsides into the soft structures beyond. As the finger is passed from without in, it comes upon a sharp declivity at the edge of the crateriform enclosure. This gives one the idea of a depressed fracture; but it will be found that the floor of the apparently sunken space lies in the natural curve of the skull. In the end there is complete levelling by absorption of the superabundant lymph, and the normal condition is restored.

**Pott's puffy tumour.** (*Vide* Necrosis, pages 284–296.)

**Scalp wounds.**—The large number of arteries in the scalp, and their very free anastomosis, explain at once the profuse bleeding, the wonderful power of repair of wounds, and the great rarity of sloughing. As the divided vessels are embedded in firm, closely-woven tissue, contraction and retraction are impeded, and hence mechanical means are usually required to stop the hæmorrhage.

**Erysipelas of the scalp** is accompanied by a good deal of œdema, which causes marked bogginess. It may lead to suppuration beneath the aponeurosis. Unless accompanied by fracture of the skull, meningitis is a rare sequel, but it may arise from spreading of the inflammation in the course of the communications between the cerebral sinuses and the external veins. The severe nervous symptoms frequently exhibited are in the majority of cases dependent on functional disturbance set up by the poison circulating in the vessels. It is thought by some that the tissues of the scalp are peculiarly liable to erysipelatous inflammation, but the explanation lies in the frequency of scalp wounds. Simple inflammatory redness and œdema may be mistaken for the specific disease.

**Tumours of the scalp.**—The most common are nævus, sebaceous cysts, and epithelioma. When a

sebaceous cyst suppurates, obstinate ulceration with the growth of fungoid granulations may simulate epithelioma, but the history of the case, and the presence of other unbroken cysts, are points that serve to clear up the diagnosis. The *pulsating swellings* comprise hernia cerebri, meningocele and encephalo-meningocele, cirroid aneurism, malignant tumours communicating or not with the interior of the skull, and, very rarely, subaponeurotic collections of pus, blood, or cerebro-spinal fluid, receiving the pulsations of the brain through the cleft of a fracture. Pulsation of the scalp without tumour is seen at the fontanelles, and very rarely where the bone is extensively absorbed in craniotabes.

## CHAPTER XXIX.

HERNIA CEREBRI—HÆMORRHAGE BETWEEN THE SKULL  
AND DURA MATER.

**Hernia cerebri** is a protrusion through an opening in the cranial walls of a soft mass, composed of highly vascular granulation tissue and softened brain substance. It essentially depends on inflammation of the meninges and cerebral cortex. It is most common after compound fracture with laceration of the dura mater. The idea that pressure of the dura mater against the edge of the inner table is sufficient of itself to set up ulceration is erroneous; for this does not happen in cases of trephining, where the operation is performed for other than depressed fracture or intracranial suppuration; or, in other words, where the soft structures are neither torn, nor weakened by acute inflammation.

The outward pressure of the brain intermits with its pulsations, and the only result is moderate thickening, the same as when a corn is produced by friction. In hernia cerebri the cerebral membranes are destroyed by inflammatory softening, and the underlying portion of brain is infiltrated with liquor sanguinis and leucocytes. New capillary blood-vessels are developed in the embryonic tissue. Inasmuch as absorption does not keep pace with exudation, and the capacity of the cranium is a constant quantity, the surplus matter escapes where there is least resistance, just as in hernia testis (page 402). The difficulty with which the local circulation is carried on accounts for strangulation and rupture of the thin-walled capillaries. The congestion and interstitial extravasation may be so great that the hernial protrusion closely resembles a

blood clot. Microscopical examination reveals vast numbers of leucocytes, dilated capillaries, apoplectic effusions, and disintegrating nerve cells. The last-named are recognised by their size and shape. As a rule, the morbid process only ceases with the death of the patient. If recovery takes place, the mass shrinks and undergoes cicatricial contraction. The fibrous tissue forms a scar and fills up the opening; sometimes it is partially or entirely converted into bone.

Should the patient survive the loss of a considerable portion of the brain, the skull adapts itself to its diminished contents by hypertrophy of the diploe and retrocession of the inner table, or by re-modelling without increase in the thickness of its walls. In the latter case the cranium loses its symmetrical outline.

Hernia cerebri pulsates synchronously with the beats of the heart.

**Hæmorrhage between the skull and dura mater.**—This is met with in many cases of injury. Separation of the dura mater is effected in the first instance by the violence of the blow, but from this cause alone it is not very extensive. It is necessarily accompanied by hæmorrhage from the torn vessels; but unless there be fracture of the skull, the bleeding is seldom profuse. In due course the extravasated blood is absorbed, or suppuration is established about the clot. When the middle meningeal artery is wounded, the consequence is far more serious, since the pressure under which the blood is poured out is sufficient to strip up the dura mater to a very considerable extent, and the bleeding may not cease until a large effusion has taken place.

The appearance of the clot when removed with the dura mater is quite characteristic. It is saucer-shaped, the outer convex surface corresponding with the concavity of the skull. The whole preparation bears a resemblance to a placenta with a portion of the

membranes attached. The size varies in most instances from two to five inches in diameter, and from half an inch to an inch and a half in thickness. The colour depends upon the length of the interval between the injury and the death of the patient, upon the degree of post-mortem change, and upon the mode of preserving the specimen. It may be dark-red, greenish-black, or jet-black.

The likeness to a melanotic malignant tumour is sometimes very striking, but it need never be mistaken for this if the uniform shape of the mass and the presence of a broad expanse of fibrous membrane (dura mater) be borne in mind.

As the large meningeal vessels are more intimately connected with the dura mater than the subjacent bone, they are carried inwards by the clot; and the greatest difficulty may be experienced in detecting the situation of a wound in the bleeding artery at the bottom of the cavity; in fact, this may be impossible, unless the aperture made by the trephine is considerably enlarged.



## CHAPTER XXX.

## INTRACRANIAL SUPPURATION.

THIS may be located : - (1) between the dura mater and the skull ; (2) in the subdural space ; (3) on the surface of the brain, beneath the arachnoid ; (4) in the substance of the brain (cerebral abscess). It has been already noted that pus may be formed as a consequence of injury separating the dura mater. This is quite certain to be the case if the bone becomes necrosed. Fracture is not essential for its occurrence. It not infrequently happens that a patient recovers from the immediate effects of the concussion, and remains free from any marked symptom for several days, or even two or three weeks ; and that then headache, fever, and local tenderness and swelling, point to the probability of intracranial suppuration.

The exact situation of the lesion as to its depth from the surface cannot be determined with certainty by the symptoms alone. If, at the bottom of a scalp-wound or abscess, the bone is dead and bare, the chances are that there is pus between the skull and dura mater, with or without more deeply-seated mischief. If the inner table has preserved its vitality, there may still be suppuration immediately beneath it ; or, the dura mater remaining adherent, localised or diffuse abscess may be found in the subdural space. In a case of gunshot injury to the skull, without fracture, I found a few drops of pus external to the dura mater, and a collection beneath it over a surface of about three inches in diameter.

Subdural suppuration is either local or general. The event turns mainly on the intensity and rapidity of the inflammatory process. Where these are

moderate in degree, the opposed surfaces at the periphery of the inflamed area are united by firm lymph, and a boundary wall is thus formed which prevents the diffusion of the pus. Abscess of the brain, the result of injury, is sometimes found without a trace of suppuration in the membranes.

Purulent effusion between the bone and dura mater in the majority of cases is due to injury, with or without fracture or necrosis. When syphilitic caries attacks the inner table of the cranial vault, a certain amount of pus exudes from the granulations, and either remains locked-up between the bone and the thickened dura mater, or escapes through a crevice by the side of a sequestrum.

In meningitis from disease of the middle ear and petro-mastoid cells, the dura mater is generally found in a sloughy condition; there can, however, be little doubt but that, previously to its perishing in this manner, suppuration to a limited extent occurred beneath it.

**Suppurative arachnitis** is extremely rare, except as a consequence of inflammation spreading from the bone and dura mater; hence a careful search should always be made for some local injury or disease, such as caries of the middle ear, or syphilitic caries. Now and then it occurs as a metastasis, from bed-sore or other form of pyæmia, but the liability to septic infection of the arachnoid is much less than is the case with the pericardium or pleura. There is sometimes increased effusion external to the arachnoid as a result of irritation from pia meningitis; but whilst the fluid may be cloudy, it never attains the purulent character, as it does in inflammation from the above-mentioned causes.

Although the pus is occasionally confined to the region of some local centre of disease, it is generally diffused over the whole or greater part of the

hemisphere. It usually escapes in some quantity on incising the dura mater; and, when that membrane is removed, the brain is seen to be covered with a thick layer of exudation, creamy or yellowish-green in colour.

**Pia-meningeal suppuration** occurring apart from head injury, tubercle, ear disease and cerebral abscess was formerly described as idiopathic. Although the exanthematous fevers were given as the probable cause on account of the not infrequent association of the two affections, there is good reason to believe that it is always pyæmic. Its relation to the exanthemata—notably, scarlet fever—is most likely secondary, *i.e.* it is engendered not by the virus of the specific disease, but by infective matter developed in a sore, *e.g.* an ulcerated throat. The fact that it has been found as a sequel of acute suppurative epiphysitis, and ulceration and sloughing about neglected vaccination pustules, confirms this view.

In tubercular meningitis there is always a considerable amount of exudation; this, in the ventricles and subarachnoid space, is cloudy and serous, and, in the meshes of the pia mater, highly fibrinous, and sometimes quite purulent. In the latter situation it appears as a sulphur-like layer beneath the arachnoid. Tubercular meningitis is most marked at the base of the brain, pyæmic at the vertex.

**Cerebral abscess** is found as a consequence of injury to the skull. It also follows acute and chronic suppuration in the middle ear and mastoid cells. In pyæmia it is comparatively rare. The symptoms depend much more upon the locality than the size of the abscess. I have known the anterior lobe of the brain to be in great measure destroyed without there being any indication of such an extensive lesion. The situation of the abscess may be suggestive of its cause. Thus, the cerebellum and the temporo-sphenoidal lobe

of the cerebrum are the usual seats of the suppuration set up by spreading of the inflammatory process from the mastoid cells and middle ear. As a rule, traumatic abscess forms beneath the part injured by a blow.

The contents of the abscess consist of exudative material and the débris of the brain substance, together with a certain amount of extravasated blood. They are prone to decomposition, and so the blood pigment is rapidly transformed into derivative compounds, and the pus variously coloured—deep-yellow, reddish-brown, or dirty-green.

The odour is often very offensive, and this, too, where there has been no communication with the external air. The walls are composed of brain tissue, infiltrated with inflammatory products and blood that has escaped from rupture of degenerated vessels. On pouring water over the surface, fine flocculi float out from the disintegrating tissues, except in cases of long standing, where there is a membranous lining to the abscess owing to the central portions of the affected area having melted away, and the peripheral become indurated.

## CHAPTER XXXI.

## MASTOID DISEASE.

DISEASE of the mastoid bone is usually a sequel of inflammatory mischief in the middle ear, though it may occur as a primary affection of tubercular origin.

It is chiefly significant by reason of its being situated in close contiguity to the cranial cavity, and thus to the lateral sinus, the meninges, and the encephalon.

The mastoid bone is occupied by pneumatic cells, the largest of which is known as the *antrum*. The latter space communicates with the upper and posterior part of the tympanic cavity by a slit-like aperture. The plate of bone separating it from the interior of the cranium is very thin.

To reach the antrum from the outer surface of the bone the spot chosen is the inferior and anterior angle formed by two bisecting lines—one drawn vertically upwards from the tip of the mastoid process, the other horizontally backwards from the upper margin of the bony external auditory meatus. In children the antrum lies at a somewhat higher level than in adults.

The depth of the antrum from the surface of the bone varies in adults from  $\frac{9}{16}$  to  $\frac{7}{8}$  inch (Birmingham). It is exceedingly shallow in infants and young children. Though gradually deepening as age advances the alteration is not very marked up to puberty. The following table records the results of Jackson Clarke's investigations.\* From this it will be seen that no definite age-ratio can be formulated.

\* *Journal of Anatomy and Physiology*, vol. xxvii. p. 412.

Age.	Thickness of the Outer Wall of the Antrum or of Accessory Air-cells.	Thickness of Bone between Antrum and Lateral Sinus.	Vertical distance above Posterior and Superior Point of Tympanic Bone from best spot for opening Antrum.	Other Features.
3 weeks . . . .	note-paper	$\frac{1}{16}$ in.	$\frac{1}{16}$ in.	
3 weeks . . . .	"	$\frac{1}{16}$ in.	$\frac{1}{16}$ in.	
1 month . . . .	"	$\frac{1}{16}$ in.	$\frac{1}{16}$ in.	
1 yr. (male) . . .	$\frac{1}{32}$ in.	$\frac{1}{4}$ in.	$\frac{1}{4}$ in.	Foramen in tympanic bone nearly completed.
1 yr. (male) . . .	—	$\frac{1}{4}$ in.	$\frac{1}{4}$ in.	
1 yr. (female) . .	—	—	$\frac{1}{4}$ in. (safe)	
1 yr. 4 mos. . . .	$\frac{1}{8}$ in.	$\frac{1}{4}$ in.	$\frac{1}{4}$ in.	Foramen in tympanic bone not quite completed.
1 $\frac{1}{2}$ yr. . . . .	$\frac{3}{8}$ in.	$\frac{1}{4}$ in.	$\frac{1}{4}$ in.	
1 yr. 10 mos. . .	$\frac{3}{8}$ in.	$\frac{1}{4}$ in.	$\frac{1}{4}$ in.	Tympanic foramen. Small spine.
2 yrs. . . . .	$\frac{3}{8}$ in.	paper	$\frac{1}{4}$ in.	Foramen in tympanic bone closed.
2 yrs. . . . .	$\frac{3}{8}$ in.	paper	$\frac{1}{4}$ in.	Foramen in tympanic bone present.
(2 yrs. 4 mos. (L.)	$\frac{1}{4}$ in.	paper	$\frac{1}{4}$ in.	Small spine. Tympanic foramen present.
(2 yrs. 4 mos. (R.)	$\frac{1}{8}$ in.	$\frac{1}{8}$ in.	$\frac{1}{4}$ in.	Spine well marked. Mastoid pneumatic throughout.
3 $\frac{1}{2}$ yrs. . . . .	$\frac{3}{8}$ in.	paper	$\frac{1}{4}$ in.	Many accessory air-cells.
4 yrs. . . . .	—	paper	$\frac{1}{4}$ in.	Many secondary air-cells. Mastoid pneumatic throughout.
5 yrs. 9 mos. . .	$\frac{1}{16}$ in. hard bone	paper	$\frac{1}{16}$ in.	Anterior $\frac{1}{3}$ of mastoid pneumatic.
6 yrs. . . . .	$\frac{1}{8}$ in. cancellous bone	paper	$\frac{1}{16}$ in.	Spine present. Anterior $\frac{1}{3}$ of mastoid pneumatic.
6 yrs. . . . .	$\frac{1}{16}$ in.	$\frac{1}{16}$ in.	$\frac{1}{16}$ in.	
6 yrs. . . . .	$\frac{1}{8}$ in.	$\frac{1}{8}$ in.	$\frac{1}{8}$ in.	Anterior part of mastoid pneumatic.
6 yrs. . . . .	$\frac{1}{8}$ in.	paper	$\frac{1}{8}$ in.	Small spine. Mastoid cancellous throughout.
8 yrs. (female) . .	$\frac{1}{8}$ in.	$\frac{3}{8}$ in.	$\frac{1}{8}$ in.	Mastoid pneumatic throughout.
9 yrs. . . . .	$\frac{1}{8}$ in.	paper	$\frac{1}{4}$ in.	Mastoid pneumatic throughout; well-marked spine.
13 yrs. . . . .	$\frac{1}{8}$ in. by secondary cells	$\frac{1}{8}$ in.	$\frac{1}{4}$ in.	Upper and anterior part of mastoid pneumatic.
16 yrs. (male) . .	$\frac{1}{4}$ in.	paper	$\frac{1}{16}$ in.	Mastoid pneumatic throughout.
20 yrs. . . . .	$\frac{1}{4}$ in.	paper	$\frac{1}{4}$ in.	

Paper signifies ordinary note paper.

The pair bracketed together were taken from the same subject.



The antrum may be enlarged by rarefying ostitis, or almost obliterated by osseous deposit—sclerosis. The escape of inflammatory products into the middle ear may be prevented or impeded by granulations, caseous matter, or new bone blocking the opening between the two cavities. Such an event is indicated by diminution or cessation of the discharge from the external meatus, together with accession or increase of pain. The consequent rise of tension within the antrum causes the disease to spread.

Suppuration in the antrum may extend in several directions. (1) It may reach the outer surface of the mastoid with or without perforation of the enclosing shell of bone. (2) It may spread to the middle fossa of the skull and attack the meninges and temporo-sphenoidal lobe of the cerebrum. (3) It may travel backwards and implicate the lateral sinus and the cerebellum. (4) It may appear in the digastric fossa, and the pus confined to the deep parts of the neck by the muscles attached to the mastoid process and their fascial expansions, may then course along the occipital artery to the carotid sheath; (5) and lastly, by way of the Glaserian fissure, the resulting abscess may point behind the ramus of the jaw, or skirting the posterior margin of the temporal muscle, gain the upper border of the zygoma. In this instance the temporo-maxillary joint is liable to become inflamed.

**Consequences of mastoid disease.**—In very acute cases such as are occasionally met with in the exanthematous fevers—notably scarlatina—the patient rapidly succumbs to *septic infection* alone, or associated with sinus phlebitis and metastatic abscesses. The intensity of the pain, and the height of the pyrexia, though as a rule very marked, are not in all cases commensurate with the degree of the local mischief, and the gravity of the general condition. Early perforation of the mastoid affords the

only chance of saving the patient from fatal blood poisoning.

Considering the close proximity of the antrum to the lateral sinus, it is noteworthy that *thrombosis* is not more frequent. When it happens the danger is extreme, for the clot being septic is almost certain to become disintegrated, in which event the débris if swept into the circulation gives rise to embolic pyæmia. In sinus phlebitis the thrombus increasing may extend to the internal jugular vein—it has been known to reach the superior vena cava.

Ballance gives the following as signs of pyæmic thrombosis of the lateral sinus—when found together he regards the group as pathognomonic: “(1) a history of purulent discharge from the ear for a period of more than a year; (2) the sudden onset of the illness, with headache, vomiting, rigor, and pain in the affected ear; (3) an oscillating temperature reaching to 103° or 105° F., and then dropping, say, below 100°; (4) vomiting, repeated day by day; (5) a second, third, or more rigors; (6) local cedema and tenderness over the mastoid, or in the course of the internal jugular vein; (7) tenderness on deep pressure at the posterior border of the mastoid and below the external occipital protuberance; (8) stiffness of the muscles of the back or side of the neck; (9) optic neuritis.” The special treatment of this complication of mastoid disease is preliminary ligation of the internal jugular vein to prevent embolism, and opening the sinus and clearing out the septic clot. If hæmorrhage occurs from the opened sinus it can be readily arrested by plugging.

**Meningitis and cerebral and cerebellar abscess** are not rare in neglected cases of mastoid disease. According to Toynbee, in children under three years the disease on spreading upwards from the antrum and cells only implicates the dura mater and

the cerebrum, whereas after that age sinus phlebitis and cerebellar abscess (Fig. 32) are most common.

It is a curious fact that not seldom a zone of apparently healthy brain tissue is found between the diseased bone and the wall of the abscess cavity. Probably, however, the intervening lymphatics and minute veins which form the paths of transit of the

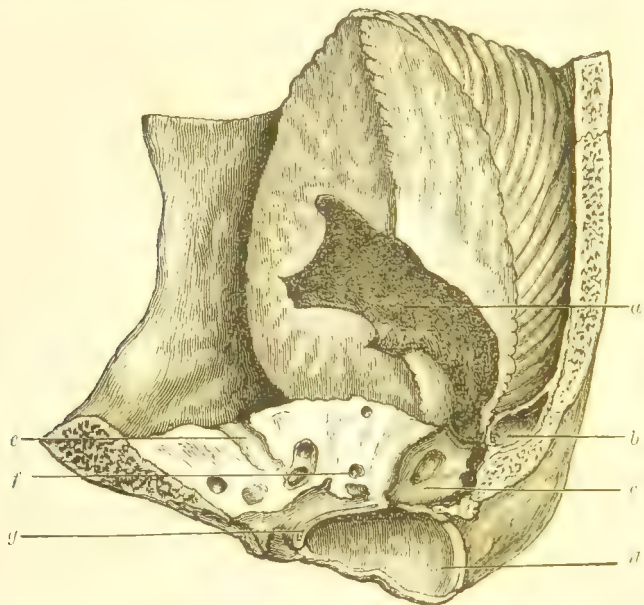


Fig. 32.—Cerebellar Abscess communicating with Mastoid Antrum. (St. Mary's Hospital Museum, No. 746.)

*a*, Abscess cavity; *b*, lateral sinus; *c*, mastoid antrum; *d*, external auditory meatus; *e*, internal auditory meatus; *f*, 7th nerve; *g*, annulus tympanicus.

septic agents are blocked by thrombi. Septic inflammation of the meninges and encephalon is necessarily fatal unless trephining is resorted to.

**Necrosis** of the petro-mastoid bone varies in extent and situation. I have seen the entire petrous bone isolated as a sequestrum. Sometimes large

portions of the outer plate of the mastoid are necrotic ; at others it is the osseous wall of the posterior and upper part of the external auditory meatus that dies. In this instance masses of granulations encroach on the passage and hinder the escape of pus. Lastly, it is not uncommon to find small sequestra in the interior of the mastoid, in the midst of granulations, pus, and caseous débris.

## CHAPTER XXXII.

## PULSATION OF THE EYE-BALL.

PULSATION of the eye-ball is accompanied by a certain amount of prominence. It is caused by (1) several varieties of aneurism, circumscribed, diffuse, ruptured, and cirroid; (2) very vascular sarcomatous growths; and (3) rarely by pulsation of the vessels in exophthalmic goitre.

Circumscribed aneurism results from direct wound of the ophthalmic artery or one of its branches, or it arises spontaneously. Cirroid aneurism (page 368) is sometimes produced by blows, or concussion of the skull, though this cause cannot always be assigned. The morbid process is probably a subacute inflammatory thickening and softening of the walls of the arteries with secondary dilatation.

In sacculated and cirroid aneurism there is usually marked bruit, and there may be distinct thrill.

## PROMINENCE OF THE EYE-BALL, EXOPHTHALMOS.

The causes of prominence of the eye-ball may be tabulated thus: (1) *Enlargements of the globe*: (a) intra-ocular tumours, *e.g.* glioma of the retina and sarcoma of the choroid and iris; (b) acute glaucoma. (2) *Orbital tumours*: (a) vascular, including aneurisms, venous naevus, and dilatation of the vessels in exophthalmic goitre; (b) solid tumours. These may arise in the orbit, or invade it from other parts, *e.g.* the maxillary antrum, and the naso-pharynx and contiguous sinuses. (3) Orbital cellulitis and abscess. (4) Absorption or depression of the orbital plate of the frontal bone in chronic hydrocephalus. (5) Paralysis of the ocular muscles, as when a gumma presses

on the third nerve. (6) Dilatation of the frontal sinus from accumulated secretion or chronic abscess. (7) Thrombosis in the cavernous sinus. (8) Hæmorrhage into the orbit, *e.g.* in fracture of the roof. (9) Congenital prominence.

Orbital cellulitis and abscess may be due to punctured wounds, fracture, syphilitic periostitis, or metastasis in pyæmia. It occasionally follows suppurative inflammation of the eyeball.

It is recognised by severe throbbing pain, and redness and œdema of the eyelids.

The continuous pressure of the cerebro-spinal fluid in chronic hydrocephalus induces atrophy of the orbital plate, and at the same time causes depression of the bone. In this way the cavity of the orbit is encroached upon, and the contents are driven forwards.

The straight and oblique muscles of the eye by their tonic contraction normally maintain a gentle compression on the globe, which keeps it steadily in its place. When several of them are paralysed, the pressure of the blood-vessels in the orbit, no longer balanced by the usual support given to the eye-ball, causes the latter slightly to advance.



## CHAPTER XXXIII.

## INFLAMMATION OF BONE.

ON account of its stability bone tissue presents a favourable field for the study of inflammation and other diseases.

Dried specimens preserve indefinitely coarse alterations in form and structure ; and by softening and staining, preparations can be made that faithfully picture the more minute changes in nutritive and formative activity.

Bone consists essentially of a rigid calcified framework, passive, but none the less important in the production of morbid states ; and of soft tissues, modified here and there according to the function they are called upon to perform. Taking a long bone, there is a layer of cartilage covering the articular ends ; a highly vascular periosteum subserving nutrition and growth ; a soft medulla accumulated in the central canal, continuous with that which forms a bed for the vessels, in the open network of the cancellous tissue and the narrow channels of the Haversian canals. All this in the foetal state is red, but at a later period the cells of the central medulla and of the cancellous tissue become loaded with fat, giving these structures a yellow appearance. Then there is, up to a certain age, a layer of developmental cartilage between epiphysis and shaft.

It may be noted that the marrow of certain bones (*e.g.* vertebræ, sternum, and ribs) retains more or less its foetal condition throughout life. For the main part the red medulla consists of embryonic cells, in which are embedded huge multinucleated corpuscles and delicate-walled blood-vessels.

The relative porosity of different parts of the same bone will to a great extent determine the issue of inflammatory and other processes; thus, in the open fretwork of the cancellous tissue, passive congestions, chronic exudations, caseation and absorption of the osseous lamellæ (the sum total of which is caries), find a more suitable nidus than in the compact bone, whose channels are so small that the blood-vessels readily become compressed, with consequent immediate death of the part (necrosis).

**Causes.**—Ostitis may be caused by traumatism, or by some morbid material in the blood irritating the medullary constituents of bones, *e.g.* of syphilis, the acute specific fevers, pyæmia, and rheumatism. In struma the carious process often follows some slight injury, though frequently no local origin can be assigned, and when it can, the destructive changes are out of proportion to the intensity of the irritation.

**Terminations.**—Ostitis may terminate (1) in resolution; (2) molar death or necrosis; (3) molecular death or caries; (4) sclerosis or condensation. The same specimen often shows the last three; *e.g.* the lower end of the femur in tubercular arthritis, where the articular surface, denuded of its cartilage, looks worm-eaten, and the cancellous structure of the epiphysis appears rarefied from absorption by granulation tissue, whilst occupying a cavity in the interior may be seen a sequestrum, and on the surface numerous stalactitic deposits of new bone. As to which shall predominate, one has to look (1) to the intensity of the inflammation; (2) to the cause, whether it be local or general; (3) to the density of the bone. The exudation may be so excessive as to lead to an acute interruption in nutrition by strangulation of the vessels, and this will occur the more readily if the spaces that contain the latter are small, as in the Haversian canals of compact bone; hence the frequency of necrosis

from suppurative inflammation of the shafts of long bones.

*Constitutional states* are a frequent cause of necrosis, as seen in the rapidly destructive inflammation of the growing part of bone in children, and in the more chronic lesions of syphilis and scrofula ; but the part that they play in the history of caries is all-important, for the same conditions that favour the onset act also against the possibility of repair ; if the vitality of the tissues is so low as to be unable to meet the physiological calls for maintenance and repair, how can it be expected to be equal to the extra task of re-construction after the ravages of disease ?

*Ceteris paribus*, the same cause (*e.g.* syphilis), varying in intensity at different times, will lead to corresponding results ; thus, an ossifying node of the tibia, caries of the skull, and necrosis of the nasal bones, may form a natural sequence in the same subject, or each may be found alone.

Again, the outcome of one source of irritation may in its turn create another, as when a sequestrum sets up a formative ostitis, or caries entails necrosis, or necrosis, caries.

## CHAPTER XXXIV.

## RAREFYING OSTITIS—CARIES.

NEXT to necrosis in order of severity is that form of destruction of bone commonly known as "caries," "molecular death," or "ulceration," in which the earthy and animal constituents are slowly disintegrated and removed. This is effected by the absorbent action of granulation tissue.

The word "caries" usually implies something more than mere inflammatory softening and porosis; it raises the question of caseation and chronic sup-puration, with their local and general consequences. If the terms caries and rarefying osteitis are considered as synonymous, several forms of inflammatory rarefaction of bone (simple, tubercular, and syphilitic) more or less distinct in their origin, course, and termination, are included in the same category. It would be well to discard the word "caries" altogether, and to describe separately each variety of rarefying osteitis.

Cornil and Ranvier maintain that rarefying osteitis is only a stage of caries, and secondary to a characteristic lesion, viz. "a fatty change destructive of the cells contained in the lacunæ."

They recognise two distinct periods in caries; "in the first the bone cells undergo fatty degeneration without there being previously the least sign of inflammation; in the second the osseous trabeculæ, killed by the death of their cellular elements, form so many foreign bodies which determine suppurative inflammation around themselves."

But they do not assign any reason for the primary degeneration of the bone corpuscles, nor explain why their death should kill the osseous trabeculæ. It

is not usual for fatty degeneration to set up inflammation, it is more often the result of it; and we are convinced that the degeneration of the bone corpuscles is the consequence of their death, and that this depends on precedent inflammation.

The heart, arteries, and cornea, when far advanced in fatty decay, do not become inflamed.

**Simple rarefying osteitis, or caries.**—When a bone is injured the blood-vessels dilate, and there is exudation of liquor sanguinis and leucocytes. Here the process may end, the *simple osteitis* subsiding, and the bone returning to its normal condition.

If the irritation is more intense, as in the case of fracture, or if it is more prolonged, as when a sequestrum is imprisoned, the inflammation becomes chronic and the exudation continuous.

The vessels, from their elongation, can only be accommodated within the rigid walls of the Haversian canals by forming loops or curves. Around these curves migratory cells accumulate, giving rise to the first appearance of granulations or buds of embryonic tissue, which enlarge and destroy the bone. Hence, instead of an even absorption, the osseous trabeculae are excavated here and there in a festooned manner.

The crypts or recesses containing osteoclasts are called Howship's lacunae.

It is supposed by some that the bone corpuscles take an active part in the process, dividing and subdividing; but most pathologists agree that the indifferent granulation-cells (osteoclasts) are merely leucocytes that have wandered from the blood-vessels and increased in size; and that the stellate bone-corpuscles show little or no sign of formative activity; that, in fact, they undergo retrograde changes, as may easily be seen where opened lacunae are setting free their degenerated contents.

There may or may not be suppuration; the event

depends mainly upon the intensity of the irritation.

A spongy bone may be honeycombed by absorption, so that it can be divided with a knife, and yet not a drop of pus be formed ; in fact, the shell of compact bone may contain little but granulation tissue.

This is what Billroth terms *caries fungosa*, or *caries non-suppurativa*. Such a case shows that the granulation tissue has sufficient vitality to survive, and that, instead of undergoing liquefaction or caseation, it may at any time (the inflammation subsiding) organise and ossify until the deposit of new bone exceeds in density the original structure.

The rarefying osteitis has passed insensibly into a sclerosing or condensing osteitis.

**Tubercular rarefying osteitis (strumous caries).**—This differs from simple traumatic rarefying osteitis, in that it is essentially due to invasion of the bone by tubercular bacilli. Local injury may start the inflammation, and thus, lowering the vitality of the part, render it a ready prey to the organisms in question.

The most common situations are the tarsus and carpus, the vertebræ and the cancellous ends of the long bones. The sternum, ribs, and mastoid bone are also frequently affected.

Some cases are so slight that the disease runs its course without suppuration or caseation (Fig. 33). The granulation tissue which fills the Haversian canals and medullary spaces, and eats out the bony trabeculae, is exuberant, semi-gelatinous, and firm, and as the inflammation subsides it organises into connective tissue and bone. This is *caries fungosa* (fungating osteitis).

But far oftener the granulation tissue breaks down ; and the pus and débris collect in an abscess cavity in the interior of the bone. The walls of the abscess are



composed of inflamed disintegrating bone, and are lined with degenerating granulation tissue.

The abscesses may remain closed indefinitely, but, as a rule, they open into a contiguous joint, or externally. This is one way in which white swelling, or strumous arthritis, commences.

Now and then the granulation tissue undergoes fatty degeneration and caseation without suppuration.



Fig. 33. Rarefying Fungous Ostitis, from a case of Strumous Dactylitis. The inflammation was very chronic. There is no caseation, but here and there an attempt at organisation.

*a*, Bone undergoing absorption by the granulation tissue; *b*; *c*, granulation bud that has perforated the osseous lamellæ; *d*, blood-vessel. The bone-corpuscles show no signs of segmentation.

The disease may be divided into three stages: (1) that of congestion; the bone is of a deep red or violet hue, giving one the idea of extravasation. This stands in marked contrast with the surrounding pinkish-yellow colour of the healthy medulla; (2) the growth

of soft vascular granulation tissue and absorption of the osseous trabeculae; (3) degeneration and softening of the neoplasia, and pus formation.

The bone-corpuscles wither and break up into fat molecules. The fat cells are destroyed. Cessation of the inflammatory products results from an inherent low vitality, and strangulation and thrombosis of the vessels. When the disease is very rapid, the cancellous spaces and Haversian canals are filled with pus.

The bone may be absorbed in such a manner that a portion is isolated from the rest by a zone of granulation tissue; then it usually dies (*caries necrotica*), but it may retain its vascular connection, and survive as a "living sequestrum."

A section through a carious bone (*e.g.* the head of the tibia) often shows the various stages of the morbid process; simple congestion at the periphery; next to this, a tract of softened bone infiltrated with a pinkish-grey soft gelatinous material; and then a pultaceous collection of pus and caseous debris, and in its midst a sequestrum.

In central caries the surface of the bone varies; it may be simply thickened from deposit of new bone, or it may be worm-eaten for some distance, from extension of the rarefying osteitis; but even then osteophytes are thrown out at the borders. Scattered tubercles may be met with at the periphery of the primary lesion.

**Superficial rarefying osteitis, or caries,** is due to the same causes as the central variety. The periosteum and superficial part of the bone are attacked at the same time. The subperiosteal medulla is converted into a red pulp, which extends into the Haversian canals of the compact bone. The osseous trabeculae are thinned, so that a probe can be driven through them. The further progress of the case depends upon the intensity of the initial irritation,

and the state of health of the patient. It may pass at once into sclerosing reparative osteitis, or proceed to suppuration and caseation. When a periosteal abscess is opened, the bone beneath, unless necrosed, is found to be more or less porous. But most cases of superficial caries are subacute, since acute exudation strangulates the vessels in the narrow Haversian canals of compact bone, so that it necroses.

Superficial caries from injury may occur in any bone. When of strumous origin it is found in the same situations as central caries. It is not uncommon in the malar bone, about the lachrymal sac, and in the mastoid process. Syphilitic caries has a special proclivity for the cranial vault, a place where strumous caries is rarely seen.

**Nature of the discharge in caries.**—It is purulent, sero-purulent, or curdy. The liquor puris holds *in suspension* caseous flakes, pus cells, granular debris, and crumbs of necrosed bone set free by the melting down of the granulation tissue; in *solution*, lactic acid and the usual alkaline, earthy, and organic constituents. There is an excess of lime salts.

**Discrete miliary tubercles in bone.**—Reference is here made to small, hard nodules, composed of groups of cells, set in a granular, homogeneous, or fibrillated matrix. The vessels of the medullary spaces and Haversian canals are obliterated by the growth, which is extravascular. This takes place before the neoplasia has time to absorb the bone, hence those portions embedded in the tubercles do not appear notched, as in rarefying osteitis.

The deposits form part of a general miliary tuberculosis, or they are the sole evidence of the disease. In a later stage the primary tubercles may be lost to sight owing to their fusion, and to being embedded in the products of rarefying osteitis. The true nature of the case, however, is still manifest by the appearance

of fresh tubercles at the periphery. In many cases of tubercular disease of bone, no miliary tubercles can be found. Their absence is explained by the early onset and rapid progress of rarefying osteitis after the invasion by the bacilli.

**Caries of the spine.**—Pott's disease is usually of strumous origin. It is often started by a blow or strain. It is essentially a rarefying osteitis of the cancellous tissue of the vertebræ. It rarely, if ever, begins in the intervertebral discs. Whilst the bodies of the vertebræ are being absorbed, an osteoplastic osteitis is taking place about the neural arches. This is a conservative process, for it prevents sudden dislocation, and so saves the spinal cord from being crushed.

As the disease is more extensive at the anterior parts of the bodies, the excavated bones fall together, hence the prominent spines and curvature.

**Appearance of the bones.**—Compare them with the regular, circumscribed excavations from the pressure of an aneurism (Fig. 8), or an encapsuled tumour, and with a spine eaten into by an infiltrating sarcoma. A carious spine in the *fresh state* is quite soft, so that a probe can be driven into it. The enlarged medullary spaces are filled with a confused mass of granulation tissue, caseous matter, and pus. In a *dried unmacerated* specimen the desiccated débris lying in the ragged hollows looks like half-set mortar. *By maceration* all this is removed, and then the open fretwork of rarefied bone gives a rough representation of the festooned outline of microscopical fragments (Fig. 34). Caseous matter on the surface of the vertebræ, the result of disease, may be mistaken for *adipocere*, derived from incomplete maceration.

Then, again, there may be an abscess by the side of the spine.

In sarcoma the bone is healthy close up to the

margin of the growth ; and there are no firm caseous patches in the interstices of the cancellous tissue.

**Fixity of the spine.**—The rigidity during life is due (*a*) to loss of the elastic discs, (*b*) ankylosis,



Fig. 34.—Caries of the Vertebra, with Angular Curvature of the Spine (One-half natural size.)

*a*, Body of vertebra, eroded and rarefied by granulation tissue; *b*, bodies of two vertebrae in which sclerosing osteitis has succeeded to carious rarefaction; *c*, neural arches ankylosed. Many intervertebral discs have been removed by absorption, and the bodies of the corresponding vertebrae fused together.

and (*c*) reflex spastic contraction of the spinal muscles.

**State of the spinal cord.**—The cord usually escapes even when the disease of the bones is far



advanced. It may be compressed by the displaced vertebræ; or the inflammation may spread to the membranes and set up a localised sclerosis of the cord; or serous effusion into the theca may compress the cord. As the motor columns lie near the bodies of the vertebræ, paralysis of motion comes on earlier, is more marked, and often occurs without paralysis of sensation.

The **curvature of the spine** (kyphosis) is angular, for the disease is mostly confined to a few vertebræ. In rickets and chronic rheumatic disease (spondylitis deformans) the curves are uniform.

**Spinal abscesses** (retropharyngeal, mediastinal, psoas, lumbar, etc.) are often very large. They may dry up, leaving a cheesy residuum. At a later period suppuration may start afresh at the seat of the previous disease—residual abscess. (*Vide* Chronic abscess, page 22.)

**Process of cure.**—As the disease subsides osteoplastic ostitis succeeds the caries, and finally the eroded vertebræ are fused into a mass of bone much denser than the original cancellous tissue. Less commonly, bars are seen to bridge over the hollows caused by absorption, which are not entirely filled in.

An anchylosed carious spine differs from united fracture of the bodies of vertebræ, in that the intervertebral discs are to a great extent destroyed, whilst in fracture they remain quite healthy close up to the circumscribed compact ossified callus (Figs. 34 and 50).

**Atlo-axoid disease.**—One vertebra only may be affected, but more commonly both are involved, and the disease may spread to the base of the skull. The situation of the caries in the region of the upper part of the cord explains the high rate of mortality in this form of spinal affection (Fig. 35).

Death may result from (1) Sudden dislocation of the atlas owing to destruction of the odontoid process



or its ligaments. In this case the cord is crushed, as in fracture of the spine. (2) Bursting of an abscess, the pus entering the air-passages and causing suffocation. This is very rare. (3) Transverse myelitis. (4) Compression of the cord by inflammatory exudation in the spinal canal, or within the theca.

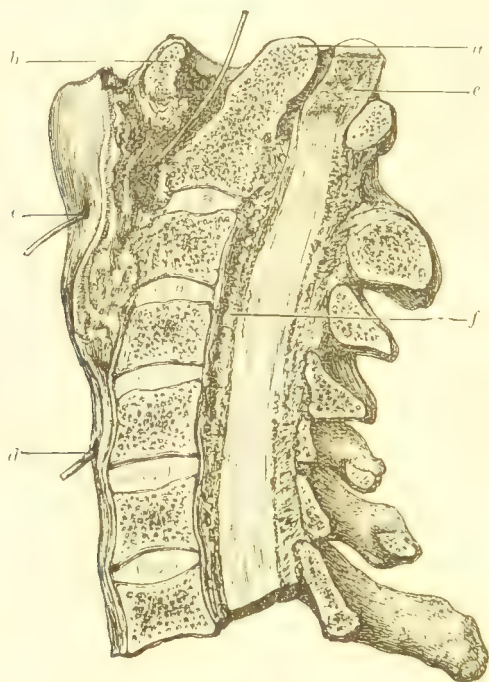


Fig. 35.—Atlo-axoid Disease. (St. Mary's Hospital Museum.)

*a*, Odontoid process; *b*, anterior arch of Atlas; *c*, opening of retro-pharyngeal abscess; *d*, opening of abscess connected with 3rd, 4th, and 5th vertebrae; *e*, cord disorganised by pressure and inflammation; *f*, spinal canal, showing extension of the disease downwards. Lymph on both surfaces of dura mater.

When suppuration occurs, the pus usually collects beneath the posterior wall of the pharynx. The abscess may open in this situation, or it may extend (1) outwards to the angle of the jaw, (2) backwards

between the transverse processes to the nape of the neck, (3) downwards and outwards to the posterior triangle, or (4) downwards into the thorax. In the last event it may perforate the intercostal muscles and form one variety of subpectoral abscess. Persistent occipital headache, especially in children, should arouse suspicion of early atlo-axoid disease.

### **Syphilitic caries and necrosis.**

— Syphilitic caries affects principally the tibia, cranium, nasal bones, palatine arch, and sternum. It begins as a periostitis and superficial otitis, or as a gumma in the substance of the bone. The osseous trabeculae are absorbed in the same manner as in simple rarefying otitis. In



Fig. 36.—Syphilitic Disease of the Cranium.

*a*, Ossified node; *b*, sequestrum; *c*, carious surface of sequestrum; *d*, apertures formed by detachment of sequestra.

the cranial vault there is frequently a number of these deposits, which are slowly absorbed, either spontaneously or as the result of treatment. The loss of substance may not be made good, so that permanent depressions are left in the outer table, and these are increased in depth by the heaping-up of new bone around them. But there is a more formidable form, in which the caries spreads over a wide surface,

and gives it a peculiar, worm-eaten appearance. Like the nodular form of the disease, it may occur with or without suppuration. It sometimes extends through the entire thickness of the bone, so that the inner plate is as irregular as the outer.

When the inflammation is more intense the vessels are obliterated to such an extent that the affected portion necroses. Fig. 36 shows a carious sequestrum. It is surrounded by a narrow trench, which in the recent state was bounded externally by granulation tissue springing from the living bone. According to Virchow, these sequestra are invariably the consequence of acute strangulation of the vessels. Cornil and Ranvier maintain that they sometimes result from an excessive interstitial deposit of new bone, which goes on until the Haversian canals are completely filled. It seems improbable that osteoplastic osteitis should alone cause necrosis, that it conduces to it by narrowing the vascular channels is obvious.

Total necrosis of the diaphysis of a bone as the result of syphilis is very rare. Figs. 42 and 43 represent two tibiae from the same leg. The original shaft (A) separated spontaneously after necrosis. It was replaced by a new one (B). The patient, a woman æt. 22, died from acute yellow atrophy of the liver ten years later.

**Necrosis contrasted with caries.**—1. *The part affected.*—Caries is more common in cancellous tissue, necrosis in compact. The blood-vessels are better supported in compact bone, and so less liable to passive congestion; but from the narrowness of the canals they are quickly strangulated by the pressure of the exudation, and so the bone is rapidly and completely deprived of its vitality. In cancellous bone there is room for dilatation and exudation, without their causing a sudden stasis in the vessels. Moreover, as the inflammation is less intense, more

time is given for the enlargement of the vascular spaces by absorption of the bone.

2. *Result of probing.*—In necrosis the probe is suddenly arrested by striking against hard bone, and without necessarily giving rise to pain ; whereas in caries it can be felt to pass through soft inflamed bone, which is very sensitive.

3. *Nature of the discharge.*—In necrosis the discharge is mostly purulent ; in caries it is more watery or serous, and contains a greater amount of lactic acid.

4. *Granulations along the sinus and at its orifice.*—In necrosis they are comparatively healthy, often fungous and florid. In caries they are small ; or large, pale, and œdematous. But they are subject to so much variation that little reliance can be placed upon them as evidence.

5. *Cause.*—The more acute the cause, whether it be a local injury, or constitutional state such as an acute specific fever, the more likely is the inflammation to end in necrosis. In scrofula, caries is more common than necrosis.

In syphilis both necrosis and caries are frequent.

## CHAPTER XXXV.

## OSTEOPLASTIC OR FORMATIVE OSTITIS AND PERIOSTITIS.

It has been shown that inflammation of bone, according to its severity, leads to necrosis, rarefaction, or new formation, and that these results may be seen side by side in the same specimen.

The ossific deposit is either superficial or interstitial (internal).

From the intimate connection between the periosteum and subjacent bone, it is impossible for one to be affected without the other being involved sooner or later. It is true the morbid process may go on in the periosteum for some time without appreciable alteration in the bone.

Acute inflammation ends in resolution, suppuration, necrosis, or it becomes chronic; chronic leads to caseation, cold abscess, caries, or the formation of osteophytes alone.

**Chronic osteoplastic ostitis and periostitis** occur under many conditions. They may indicate a deep-seated destructive inflammation, as central caries; or arise from injury, or the irritation of a chronic ulcer, or some constitutional state such as syphilis, rheumatism, etc.

**Anatomical changes.**—The periosteum is at first succulent and thicker and redder than natural. The fibrous and medullary layers lose their distinctive outline. The whole is infiltrated with exudation cells, and can be easily separated from the bone.

The connective tissue around the blood-vessels in the Haversian canals of the compact bone is increased in like manner. It is probable that in most cases rarefaction of the bone takes place to a slight extent. As organisation advances, the periosteum acquires a

greater density, it has fewer cells, and these are more elongated. Lime salts are deposited around the vessels as they pass from the bone to the periosteum, and the embryonic cells are imprisoned in the matrix as bone corpuscles. The new osseous trabeculae stand at right angles to the surface of the old bone. Whilst there is continuity of structure between the osteophytes and subjacent bone, the former can be easily detached at this early period.

The new bone is at the first quite porous, and it may remain so; but in some cases the deposit does not cease until it is as dense as ivory.

**Varieties of osteophytes.**—1. *In an unreduced dislocation* a false joint is constructed, and to give security to this, new bone is deposited where the pressure is intermittent, *i.e.* where it is exerted only in certain positions of the more movable bone. Thus, in subspinous dislocation of the shoulder the buttress of support is situated on the dorsum of the scapula. Here it is seen as an isolated compact mass, looking as though it had been soldered on to the healthy surface, there being no sign of caries or necrosis. Such an osteophyte is coarsely furrowed and convex on one side, somewhat smooth and concave on the other. Its isolation at once distinguishes it from the osteophytes of chronic rheumatic arthritis.

2. *In chronic rheumatic arthritis* each bone entering into the formation of a joint has rounded outgrowths about the whole articular margin. They appear as if they had once been in a softer condition, and had become gradually solidified, for they droop like melted tallow or wax that has congealed on cooling. They really grow in great measure from cartilage. The articular surfaces are eburnated from the same sclerosing ostitis (Fig. 48).

3. *In strumous arthritis and caries* the extent of bony deposit fluctuates widely in different preparations;



it may amount to a mere roughening of the surface, or form closely-packed hard craggy masses, that bear the same contrast to the osteophytes of dry arthritis as do stalactitic rocks to water-beaten boulders.

The articular surface is usually porous from caries (Fig. 16).

4. *In chronic ulcers* of the soft parts, lying near to a bone (e.g. varicose ulcer of the leg), the deposit, from irritation of the periosteum, forms a flattish mass, which generally has a very defined steep margin. The surface is porous, but fairly uniform.

5. *In rickets*, when the curvature of a long bone is very decided, the concavity is occupied by a beam of compact bone thrown out to support the arch (Fig. 17).

6. *In ossifying peripheral sarcomas of bone*, the new bone radiates from the surface of attachment in the shape of



Fig. 37.—Acicular Outgrowths of Bone in the Base of a Subperiosteal Sarcoma springing from the Epiphysis of a Long Bone.

long delicate needle- or spray-like processes, that bear the impress of a centrifugal growth so characteristic of these tumours. They are, in fact, casts of the intervacular spaces (Fig. 37).

7. *Bony tumours.* (*Vide* Osteomata, pages 513-22.)

8. *In locomotor ataxia.* (*Vide* Trophic lesions, pages 152-59.)

**Nodes.**—A node is a localised inflammatory thickening of bone. At first it consists merely of a soft vascular swelling of the periosteum. This may disappear, or break down and ulcerate, or ossify. It is very tender on pressure, and usually gives rise to tensive aching pain (*vide* Pain, pages 24–26), especially at night, when increased warmth causes a fluxion to the part, and the patient's attention is concentrated more upon himself.

**Varieties of nodes.**—Nodes are classified as to their anatomical condition, ossified, carious, etc.; or as to their cause. The latter basis is here employed.

(1) *Simple nodes* are due to injury, and hence they are most common in the bones that are least protected, *e.g.* the tibia.

(2) *Syphilitic nodes* are met with both in acquired and congenital syphilis. In the acquired form they may be located on any of the bones, but the seats of election are the tibiæ, cranium, clavicles, and ulnæ. As a rule, they ossify; but, if left untreated, they frequently break down. They belong to the tertiary stage of syphilis, and are really gummata.

They are simulated by erythema nodosum; but the latter disease affects younger people, generally females; it is more transient; it is usually symmetrical, the part is movable over the bone, and it never suppurates. (For description of congenital syphilitic nodes, *vide* Osseous lesions in congenital syphilis, pages 279–283.)

(3) *Rheumatic nodes* may be attached to the bones, but more frequently they are simply fibrous thickenings of fasciæ.

(4) *Typhoid nodes.* During an attack of typhoid fever, or whilst convalescence is being established, the periosteum may inflame over localised areas. These nodes, like most others, are generally situated on the tibiæ. Their tendency is to disappear; they rarely

suppurate, and still more rarely end in necrosis. In a case I was called to at the London Fever Hospital, the patient, a young adult male convalescent from typhoid, was the subject of several recent nodes, which disappeared spontaneously after a short time. On the same bones (tibiae) were old ossified syphilitic thickenings.

**Internal or diffuse osteoplastic osteitis.**

*Synonyms.*—Diffuse hypertrophy, condensing osteitis, sclerosis ossium.

*Causes.*—In many cases no cause can be ascertained. In some there is a history of syphilis, in others tuberculosis. I once had under observation two patients, the subjects of congenital syphilis, in whom the hypertrophy of bone was well marked. In one, a male aged eighteen, there was characteristic notching of the teeth; the *tibiae* were enlarged throughout, much elongated, and curved forwards; the osseous lesion had existed for years. At the age of twenty, interstitial keratitis first made its appearance. The mother of this patient was under treatment at the same time for syphilitic *nodes* of the tibiae. The second case was that of a woman aged twenty-two; the teeth were notched, and the cornea nebulous from past keratitis; the *left radius* was uniformly rounded, thickened, elongated, and bent in a strong outward curve. There can be no doubt but that these two patients suffered from diffuse interstitial osteitis: for periostitis alone, whilst it would account for the circumferential enlargement, would not explain the marked elongation.

**Ostitis deformans.**—We are indebted to Sir James Paget for a description of this remarkable affection of the osseous system, which is usually found in persons past the prime of life. In most cases there is an absence of specific history, and the malady runs its course unchecked by antisyphilitic treatment. It

attacks several bones at the same time or in sequence ; those of the skull and lower extremities are particularly liable to suffer. From the multiplicity of the lesions, the disease may be considered as one of the osseous system in general, and not a chance affection of one or more bones.

The long bones—*e.g.* the femora—are curved in spite of the increase in thickness ; this can be explained by the fact that during the early stages the bone is rarefied and weakened by the inflammation. At the end of the process, however, there is marked increase in density, and the medullary cavity of the hollow bones is obliterated. The surface is roughened from periosteal deposit (Fig. 18).

The skull may be an inch or more in thickness ; there is general hypertrophy of the tables, with filling in of the diploë and obliteration of the sutures.

The disease is very chronic, lasting through many years, and during this time the general health may not suffer. It is noteworthy that, after the lapse of years in several instances, the bones have been found to be the seat of malignant tumour (sarcoma), either as a solitary growth, or scattered here and there. Butlin's observations show that the initial lesion is inflammatory.

**Hypertrophy of the facial bones, pelvis, etc.**—Billroth says, " In such cases the bony deposits are spongy, puffed, nodular, so that the bone acquires a resemblance to skin affected with elephantiasis." The etiology is quite obscure. (*Vide* chap. xx.)

**Senile thickening of the skull.**—In old people the cranial bones are often thicker and denser than normal ; the diploë is replaced by solid bone. It is probably of the nature of a nutritive degeneration (like enlargement of the prostate), with increased formative activity. Rickets sometimes causes great thickening of the skull.

## CHAPTER XXXVI.

ACUTE SUPPURATIVE PERIOSTITIS, OSTITIS, AND  
OSTEOMYELITIS.

WHEN it is remembered that there is direct structural continuity between the periosteum and the medulla contained in the Haversian canals of compact and cancellous bone, and, in the case of the hollow bones, that of the central cavity as well, it is no matter of surprise that acute inflammation beginning in one or other of these situations should spread through the entire tract.

Acute suppurative periostitis cannot occur without ostitis; osteomyelitis almost to a certainty entails periostitis; yet, for clinical purposes, it is found convenient to treat of them separately.

**Acute periostitis and periosteal abscess.**

—The periosteum consists of two layers. The superficial is composed of white fibres, and is the means of supporting the blood-vessels for the bone. The deeper is made up chiefly of elastic fibres, with osteoblasts in the part next the bone. In it is a fine vascular network. It is in this deeper layer that acute periostitis usually begins. At first it is swollen and red from vascular congestion; this is quickly followed by a rapid exudation of leucocytes and liquor sanguinis, so that the membrane is converted into a purplish pulp. The formed elements melt away, and the debris mingles with the pus. Thus we see that the periosteum is destroyed by the inflammatory process, which meanwhile has spread to the surrounding soft parts (muscle, cellular tissue, skin, etc.), and has made them highly œdematous.

The abscess is bounded below by bare bone, and

superficially by a layer of inflamed tissue. At the same time, the superficial portion of the bone is infiltrated with leucocytes, and part of the exudation, collecting on the surface, raises the periosteum ere the fibrous layer has completely softened (sub-periosteal abscess). This is the explanation of the so-called "burrowing of pus and stripping-off of the periosteum."

When the bone is deeply seated, an enormous accumulation of pus may form around it. When superficial (*e.g.* the anterior part of the tibia) the abscess usually points quite early. In some cases the process is so acute that beads of pus may be seen on cutting into the inflamed part within thirty-six hours from the onset of the disease.

When due to injury, the abscess is usually localised. When caused by infective organisms it is often very diffuse; this is notably the case in children.

*Terminations.*—The periosteum may be destroyed over a considerable area, and yet the subjacent bone survive; but the cutting off of the blood supply from the surface, and the great tension on the vessels in the compact tissue, place it in imminent peril of death.

Early and free incisions by draining off the exudation and unloading the engorged vessels may prevent suppuration.

Even when pus has formed, resolution is sometimes rapid and complete after the bursting or opening of the abscess. In such cases the inflammatory tissue that bounded the purulent collection superficially applies itself to the bone and organises into a new periosteum.

There is probably in all cases a certain amount of new bone deposited on the surface of the old and in the Haversian canals of the compact tissue.

Necrosis is a frequent result.

**Acute osteomyelitis.** — Acute osteomyelitis occurs as (1) a diffuse inflammation of the soft



tissue of bone, such as we see in a compound fracture, or after amputation where the wound has become *septic*; or (2) it is set up by extension of acute inflammation from a contiguous joint; or (3) it arises as a primary *infective* process. This variety commonly follows an injury which acts mainly by lowering the vitality of the part—*i.e.* making it more vulnerable—and thus rendering it a more ready prey to the essential agent—a micro-organism. If the specific infective material be injected into the veins of two animals, the one healthy, the other with a recent simple fracture, the former will suffer merely from constitutional disturbance, often only temporarily; the latter will develop acute infective osteomyelitis at the seat of injury.

As the exudation is pent up under high pressure and there is no possibility of an early spontaneous evacuation, necrosis is almost certain to follow.

The patient often dies before the sequestrum is loosened.

The inflammation may lapse into a chronic state, an exit for the pus being provided by operation, or the opening up of channels in the bone by rarefying ostitis.

**Osteophlebitis**, or **osteo-thrombosis**, is a concomitant of osteomyelitis, whether pathological or traumatic. The coagula are very liable to disintegrate and break up into emboli, for they are steeped in a highly irritative or infective fluid. Moreover, as the veins lie in rigid canals, to the walls of which they are adherent, they are unable to collapse when divided or torn.

## CHAPTER XXXVII.

## OSSEOUS LESIONS IN CONGENITAL SYPHILIS.

AFFECTIONS of the osseous system in congenital syphilis stand midway between those of the skin and the viscera in order of frequency.

Our knowledge of the subject has been mainly derived from the researches of Wegner and Parrot abroad, and Drs. Barlow and Lees in England.\*

In one way or another every part of the skeleton is liable to be diseased, but the points of greatest interest are centred in the *long bones*, the *cranium*, and the *teeth*.

**Cranial bones.**—The lesions here are of two kinds : (1) atrophic ; (2) osteophytic.

Localised wasting of the cranial vertex is met with in the parts most subject to pressure from decubitus, and it is all the more marked if at the same time there is an increase of fluid in the cerebral ventricles. The usual situation is the parietal bone behind the eminence, but it has been observed in the occipital and squamous bones. The bone is gradually absorbed until nothing is left but a thin plate, or membrane, of parchment-like consistence, which readily yields under the pressure of the finger. The same condition occurs in rickets, but not so frequently as in congenital syphilis. The absorption leads to a well-marked depression of the inner surface of the bone.

It is a case of atrophy from continued pressure acting upon a tissue preternaturally soft and weak. The process is designated *craniotabes*. It is found in very young infants. M. Parrot has described

\* An admirable account of the lesions is contained in the Transactions of the Pathological Society, vol. xxx.

another form of atrophy, which he terms "gelatiniform." As the name denotes, it consists of conversion of the bone into a soft material. It begins beneath the pericranium, but rarely spreads as far as the dura mater. It cannot be diagnosed during life.

*Parrot's osteophytes or nodes* are subperiosteal deposits of new bone

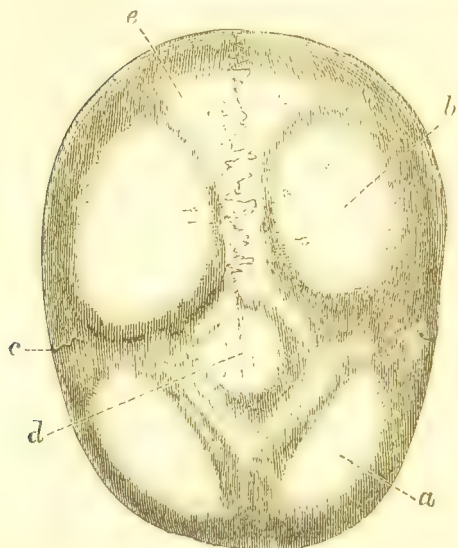


Fig. 38.—View of Outer Surface of Cranium affected with Congenital Syphilis.

*a* and *b* point respectively to a frontal and parietal node or boss. These nodes are composed of bone of a coarser texture than that which bridges over the anterior fontanelle, *d*; *c*, fronto-parietal suture; throughout the greater part of its extent it is obliterated by ossific deposit; *e*, parietal eminence. The natiform nodes bounding the anterior fontanelle are somewhat too sharply defined in the diagram. The preparation is in the Museum of the Hospital for Sick Children, Great Ormond Street, and is represented here by permission of Dr. D. Lees.

posits of new bone (Fig. 38). They are situated upon the four processes that bound the anterior fontanelle, to which they give a natiform appearance. Other deposits are usually found upon the parietal bones, and chiefly along the coronal and sagittal sutures. It will be seen that they affect neither the frontal nor parietal eminences, nor the sites of craniotabes, or only in very severe cases. The new bone is very vascular and porous when first formed. It consists of a series of trabeculae, between which are

spaces filled with a pulpy marrow. The vessels run at right angles to the surface of the skull.

The bony eminences may attain a thickness of more than half an inch at their centres.

The fronto-parietal sutures are sometimes obliterated by an extension of ossification from one bone to the other.

These osteophytes are characteristic of congenital syphilis. In the majority of cases they form between the sixth and twelfth month. Ulcerating nodes, though common in acquired syphilis, are excessively rare in congenital; Barlow records one such case.

The **long bones**.—As in the cranium, the disease shows itself in two forms, atrophic and productive.

*The atrophic changes* are seen in the growing layer of the tissue on the diaphyseal side of the epiphyseal cartilage. It is sometimes described as an osteochondritis, but it appears to consist essentially of a deviation from normal development. The inflammatory changes are secondary, and dependent upon injury.

There are two distinct changes: (1) an excessive deposit of lime-salts in the cartilage matrix and capsules, and imprisonment of the cartilage cells. This, whilst it increases the brittleness of the part, puts a stop to the ossification; (2) an overgrowth of gelatinous medulla or spongeoid tissue in the ossiform layer (that next the cartilage); this absorbs the pre-formed bone, and occasionally ends in complete separation of the epiphysis. The fragility due to calcification and the softening caused by "gelatiniform atrophy" render the bone liable to bend or break on the slightest violence. Then the irritation caused by the injury sets up a veritable inflammation, which may end, though rarely, in suppuration. If the child survives the cachexia of the disease, the epiphyseal lesion subsides, and ossification goes on in the normal way.

When the morbid process is in progress the limb hangs helplessly from the trunk. Parrot calls this

condition "syphilitic pseudo-paralysis," to distinguish it from essential or infantile paralysis of nervous origin, in which there is actual powerlessness of the muscles.

*Osteophytes* form, as a rule, after the sixth month of extra-uterine life. The bones most frequently affected are the tibia and humerus. I have seen symmetrical osteophytes on the upper ends of the ulnæ. They consist of highly vascular spongy bone, the trabeculae of which stand perpendicularly to the axis of the limb. They are usually constructed of a number of superposed lamellæ, united by narrow bars or columns, embedded in a soft medulla. Sometimes the osteophytes are quite soft, and are made up chiefly of fibro-vascular tissue. Between the two forms there is every variety. The tibial, ulnar, and radial osteophytes are mostly situated at the upper epiphyses; the humeral at the lower. In the scapula they are found in the spinous fossæ; in the hip, in the external iliac fossa.

**Maxillæ and teeth.**—The characters of the teeth described by Hutchinson consist of malformation and defective structure. Those most commonly affected are the upper *permanent* central incisors. Instead of the borders of the cutting edges being in contact, they are separated by a gap. There is a central indentation or notch in the place that should be occupied by the middle tubercle. The lateral incisors are sometimes pegged, and the canines (upper and lower) more pointed than natural. The *milk teeth* are subject to early decay and premature shedding. The date of eruption is not deferred, as it is in rickets (Eustace Smith). Hutchinson attributes the mal-development to specific stomatitis, which affects the gums, periosteum, and bone in infancy.

**The nose.**—Flatness of the bridge of the nose is frequently met with as the result of congenital syphilis.

though it is not characteristic of it, since scrofula induces a like result. The faulty construction of the bridge is due to periostitis, which usually goes on to caries and commonly to necrosis. The affected bones are smaller than natural, since the inflammatory lesion checks their development. As a secondary consequence the expansion of the sphenoidal sinuses is defective, and thus the mechanism by which the vomer is carried forwards and the septum nasi raised into position is limited in its action.

In the tertiary period serious lesions are met with; namely, caries and necrosis of the nasal and other bones, hypertrophy of the long bones from diffuse interstitial osteoplastic ostitis, and localised thickenings from osteo-periostitis (nodes).



## CHAPTER XXXVIII.

## NECROSIS.

NECROSIS happens when the circulation in a portion of bone is permanently arrested, from injury or disease. In the former case, if many vessels be ruptured by violent concussion or fracture, the current through them is arrested by thrombosis in the torn ends, and by the pressure of extravasated blood. In fracture, too, with considerable displacement of the fragments, and especially if there be much splintering, portions may be entirely denuded of periosteum. Should the bone survive these accidents, the circulation has to cope with the obstruction from inflammatory exudation, so that it is not surprising that necrosis is often of traumatic origin.

Syphilitic osteitis leads to necrosis, either immediately by occlusion of the vessels from the pressure of the effusion, or more remotely through the Haversian canals becoming so narrowed by deposit of new bone that a slight increase of the inflammation suffices to obliterate the vessels within them.

When the subject of abscesses in carious epiphyses is discussed it will be shown that they often contain sequestra of considerable size, the result of acute osteitis, or of caseation of tracts of granulation tissue, with thrombosis of the vessels.

**Superficial necrosis, exfoliation.**—A good example of this is seen in injuries to the cranial vertex, where the pericranium is destroyed, with probably some slight extravasation into the diploë and external plate. Unless the dura mater be detached, the inner table escapes death, for it is plentifully supplied by the meningeal vessels. Inflammatory effusion completes

the stasis in those parts already crippled, but it must not be supposed that the inflammation is limited to the part that dies, for it gradually subsides towards the healthy bone. The layer that is continuous with the necrosed portion becomes rarefied by absorption, the granulations thrusting their buds through the walls of contiguous Haversian canals, so that the latter open into one another. Finally, the attenuated osseous laminae disappear, and the dead part is cast off (Fig. 31). These changes take place chiefly in the tissue that has retained its vitality, but that the inflammatory new formation is able to absorb dead bone is proved by the erosion of ivory pegs employed in ununited fractures. Moreover, it is not rare to find perforations of thin superficial sequestra by pink granulations—a welcome sign to the surgeon, for it tells at once of the shallow depth of the necrosis. It is this riddling of the bone that causes it to crumble down during detachment. The source of irritation being removed, the layer of florid embryonic tissue that covers the surface begins to organise. Bone is deposited around the vessels. It acquires considerable density. A new periosteum is constructed from the outer layer of granulations.

The exact method of absorption of bone is not known. Virchow believes that cells (osteoclasts), derived from proliferation of bone-corpuscles, are the active agents. Rindfleisch suggests that the blood in the congested vessels containing an excess of carbonic acid may dissolve the lime salts, forming an acid carbonate. Others suppose that lactic acid is developed, and that this, combining with the earthy base, forms soluble calcic sarco-lactate. The two latter hypotheses are improbable, for bone exposed to the action of pus for months or years loses little or none of its substance, and it retains its smoothness of surface from the time when it becomes sequestered.

Whilst denying the origin of the bone destroying cells ascribed by Virchow, I believe his view of absorption by the vital action of living matter to be quite rational. An exfoliated lamina of bone is smooth on the outer surface, where it undergoes no change of structure in the osseous framework, but it looks worm-eaten on the under surface, the indentations having been formed and occupied by vascular granulations. If vertical sections of artificially-softened bone be made during the process of separation of a sequestrum, it will be seen that the Haversian canals of the necrosed portion are empty, or contain nothing but the débris of disintegrated marrow, whilst the spaces of the living bone are filled with embryonic cells and blood-vessels in a state of active proliferation. Picrocarmine stains the osseous trabeculae yellow and the granulation tissue red, and shows a beautiful layer of demarcation which ends abruptly, and is most intense in colour next the dead bone, but gradually fades away into the living. The adjacent periosteum is infiltrated with indifferent cells, especially in the deeper part.

Pus escapes from the vessels of the granulations at the margin, and also beneath the sequestrum when this is loose.

**Necrosis of an amputation stump.**—Amputation through a long bone is a good example of compound fracture, and the wonder is that necrosis does not more frequently result, for the vessels of the medulla and periosteum are severed, and their ends compressed by extravasated blood, and necessarily plugged by clots. At the same time the vitality of the bone may be impaired from disuse or existing inflammation, both of which conditions readily allow of detachment of the periosteum if traction be made upon the flaps.

Without exception, the injury inflicted by the

operation sets up inflammatory action ; the medulla of the central canal, that contained in the substance of the bone, and the periosteum all show acute hyperæmia and exudation, and return to the embryonic state. This is followed by rarefying osteitis. In ordinary cases the inflammation does not go beyond this, but, subsiding, ends in a condensation from deposit of new bone which greatly narrows the vascular channels and fills the open end of the medullary canal. Upon the completion of these changes, atrophy from partial loss of function sets in, and the end of the bone thereby becomes conical.

The **sequestrum**.—In the event of necrosis, one or more of the hindrances to the circulation is increased ; thus, the periosteum is stripped off to such an extent that the bone cannot recover itself, or a central osteomyelitis destroys the medulla (Fig. 39). Stasis from the latter

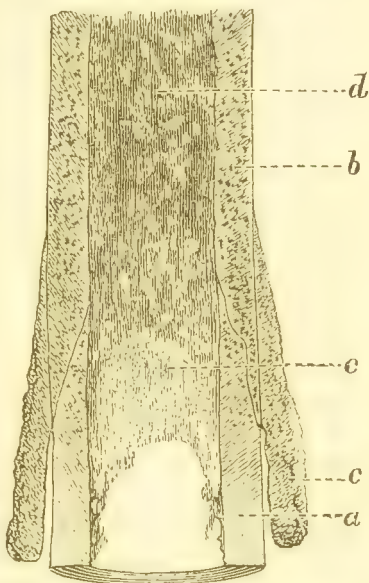


Fig. 39.—Necrosis of Femur after Amputation of Thigh.

*a*, Dead bone more extensive on medullary aspect ; *c*, new bone deposited from periosteum, adherent to *b*, the living portion of the shaft, but free from the necrosed portion, *a* : *d*, medulla deeply congested and hemorrhagic ; *c*, the same, decolorised and purulent, in process of disintegration.

cause usually exceeds that from the former, and so the necrosis is more extensive on the inner aspect, accounting for the sequestrum appearing in the shape of a truncated cone whose outer surface is excavated into shallow pits and grooves by the granulation tissue during the process of separation. Whilst the

rarefying osteitis is going on in the substance of the bone, in order to set free the dead portion, ossification is progressing in and beneath the periosteum, so that the outer surface becomes encrusted with soft spongy bone. When once the sequestrum is removed—and it usually requires an operation to effect this—the subsequent changes differ in no way from those occurring in the healing of a stump by granulation without necrosis.

These sequestra vary much in size. They may be many inches in length, and of great thickness, or so small as to break in pieces during attempts at removal. Their lower ends are sometimes smooth on the outer surface for a short distance, showing that the periosteum was destroyed to a like extent, and that the granulations left untouched the denuded bone. In other cases the outer surface is smooth, and the inner rough throughout, the loss of vitality being more of periosteal than medullary origin; it is, in fact, superficial, and not central necrosis.

Whatever the variety, the general shape is quite characteristic, the sequestra ending abruptly below with a sawn surface, tapering above. The cavities from which they are removed are never bare, but are lined by soft vascular granulation tissue, which, increasing, soon fills up the spaces and ossifies.

**Central or internal necrosis** may ensue from osteomyelitis, without a previous loss of continuity in the bone, the osteomyelitis being either primary, or forming a part of a more widespread lesion of periosteum and medulla. In the former case it begins (1) as an acute suppurative inflammation in the central canal, when it is often fatal before the sequestrum is loose; or (2) as a tubercular or infective para-epiphysitis, which spreads to the interior of the shaft.

In the tubercular variety the march of events is

less rapid, so that there is time for the inflammation to subside and the dead bone to be thrown off into the medullary canal. Relief is given to the tension by the bursting of an abscess which communicates directly with the exterior, or in a more roundabout way by the contiguous joint.

Tuberculosis being often at the root of the evil, it is not surprising that caries and joint disease complicate this form of necrosis. The medullary canal becomes filled with granulation tissue and pus, in which the sequestrum is embedded.

Outlets for the pus are made here and there by absorption of the osseous trabeculae, where the tension is greater than elsewhere. These outlets, which in the case of bone are called *cloacae* (Fig. 40), correspond to sinuses in the soft tissues.

Central can always be told from total necrosis by the outer surface of sequestrum appearing rough and worm-eaten in the former, smooth in the latter.

The encasing shell of bone is entirely of new formation in total necrosis, whereas in central necrosis it is composed of the outer portion of the original shaft, thickened by deposit of new bone.

Sequestrotomy is necessary for the liberation of the sequestrum, which, so long as it is retained, keeps up suppuration, a cause of lardaceous disease. Nature, unaided, seems content with lowering the tension to the level of chronic inflammation.

**Acute total necrosis.**—It is impossible, clinically, to draw a hard-and-fast line between so-termed partial and total necrosis; indeed, such a division is unnatural, for many of the cases complete in the pathology of the disease in question fall short of death of an entire diaphysis. The symptoms and morbid signs are, notwithstanding, so marked as to justify a special description. Stripped of its details, the history runs thus: An often unaccountable onset, rapid progress,



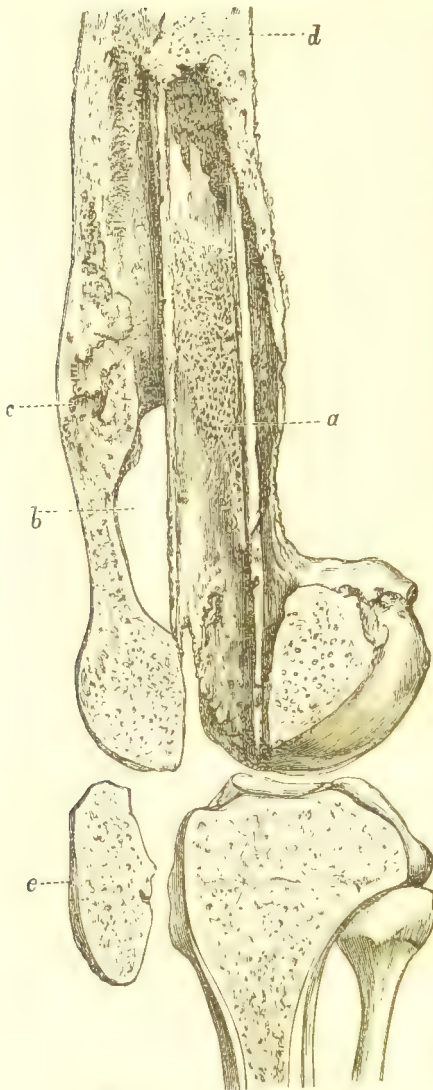


Fig. 40.—Necrosis of Femur.

The sequestrum, *a*, had been locked up for eight years, and had by its weight and friction bored a passage through the epiphysis into the knee-joint (atrophy from continuous pressure); the articular cartilages are but little affected; *b*, cloaca, through which the pus escaped from the sequestrum cavity, which in the fresh state was lined with granulation tissue; *c*, new bone; *d*, osseous deposit that has filled in the medullary canal; *e*, patella; behind this, the tibia and fibula. (Half natural size.) In this case the urine contained half albumin before amputation, only one-fifteenth by the time the stump had healed, and two years afterwards none.

tendency to end in a fatal pyæmia with secondary lesions, and liability to select the periods of childhood and youth. It is an acute infective suppurative inflammation of the growing part of bone, at one time limited to a periostitis and superficial osteitis, at another involving the destruction of the epiphyseal cartilage, and even the entire medulla.

The epiphyses themselves, as a rule, escape, or, at any rate, are not affected past recovery, although the inflammation may spread through them to the contiguous joints.

**Causes.**—Injury, acute specific diseases, such as scarlet fever and measles, and exposure to wet and cold, have been assigned as

the causes, but at most they can only be considered as predisposing agents. The disease is essentially an infective one—*i.e.* it is due to invasion of the bone by a micro-organism.

**Method of invasion.**—The seat of greatest activity in a growing bone is the layer continuous with the epiphyseal cartilage on the diaphyseal side, and next in order comes the periosteum. The physiological instability of the tissues in question renders them more liable to succumb to micro-organisms capable of setting up suppuration. Experience teaches, as would be expected, that a large number of the cases of “acute necrosis” commence as *epiphysitis* (Fig. 41). The inflammation, once established, rapidly

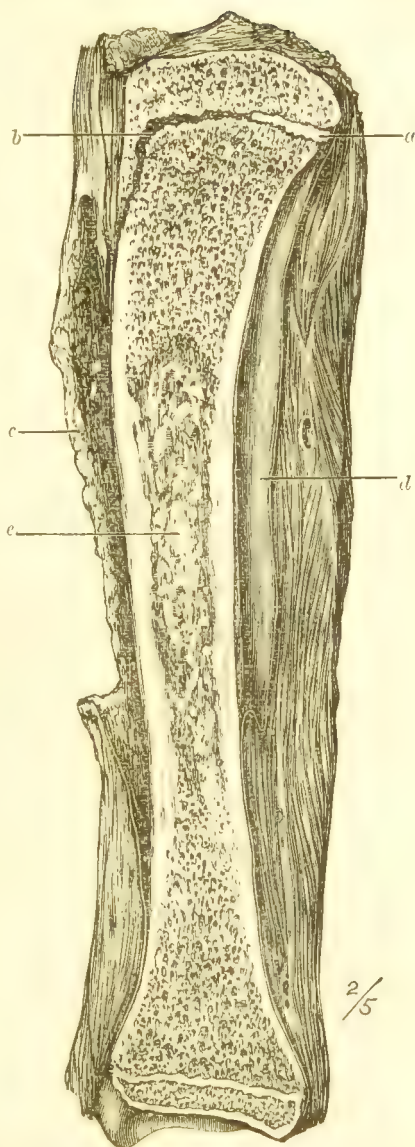


Fig. 41.—Acute Necrosis of Tibia. (From St. Mary's Hospital Museum.)  
*a*, Remains of epiphyseal cartilage; *b*, space where epiphyseal cartilage has been destroyed; *c*, irregular margin of skin which bounded a large open wound; *d*, subperiosteal abscess cavity; *e*, collection of pus in the medullary cavity.

spreads to the contiguous parts: (*a*) beneath the periosteum along the shaft of the bone, (*b*) through the bone to the medullary cavity, and (*c*) to the neighbouring joint. Should the margin of the para-epiphyseal layer be first attacked, the inflammation will invade the periosteum in preference to the medulla, or, in other words, will extend along the path of least resistance. On the other hand, osteomyelitis is more likely to ensue when the disease begins in the centre of this layer.

In some cases the process starts as a periostitis, and in others, though very rarely, as an osteomyelitis at some distance from the epiphysis—*e.g.* in the central medulla. The disease has been defined respectively as (1) infective epiphysitis, (2) periostitis, and (3) osteomyelitis; but whatever phase it may assume in the first instance, its pathological identity should not be lost sight of.

**Diagnosis.**—The severity of the constitutional symptoms and the obscurity of origin account for many of the mistakes in diagnosis. Children are sometimes brought to hospital because a supposed erysipelas or acute rheumatism has not run an expected course, the first idea of the real state of things being aroused perhaps by the evidence of fluctuation over a bone; and even this may be overlooked, the enigma being solved on the *post-mortem* table. A previously healthy girl of fourteen was treated for rheumatism, but getting rapidly worse, with high fever, widespread muscular spasm, and delirium, was taken to the medical ward of a hospital, where at first the possibility of cerebro-spinal meningitis was entertained, there being no apparent sign by which to localise the disease. After death the right clavicle was found lying in a bed of pus, and the lungs were riddled with small metastatic abscesses. It is unpardonable to mistake a case of acute necrosis for one of erysipelas or rheumatism; and even when the general disturbance caused by the

blood-poisoning is so great as to mask the local lesion, it ought to suggest a thorough examination of the bones, especially in children, in whom the disease very commonly begins as an infective epiphysitis.

The usual local signs are tensive pain and exquisite tenderness, redness of the skin, and fluctuation or bogginess of the part.

**Dissection.**---The skin and cellular tissue appear congested and highly cedematous, the periosteum is detached to a varying extent, and the bone over the same area is bathed in pus. The epiphyses may be separated from the diaphysis, and the joints healthy or inflamed; in the latter case, either by spreading of the local disease or by pyæmic metastatic infection.

If the bone be sawn vertically, the central medulla will present a deep-red colour from congestion, with here and there patches of capillary extravasation, interspersed, perhaps, with collections of pus, which are generally situated at the periphery, the canal for the nutrient artery forming a purulent tract that connects the superficial with the central suppuration.

The compact and cancellous tissue present a mottled appearance in place of the diffused pinkish-white tint of healthy bone, for the spaces are occupied by purulent exudation and dilated vessels, which are filled with dark deoxydised clots. Later, of course, the contents of these spaces disintegrate, and leave the bare osseous framework white or yellowish-white, and lustreless.

Venous thrombi within and without the bone may be softened, and ready to develop septic embolism.

If any part of the bone escape death, it becomes encrusted with ossific deposit, which forms an appreciable layer within ten days provided the tension has been relieved by incision. Should the necrosis not be total, it is the deeper portion of the bone that survives, for this receives its vascular supply more

directly from the large vessels, the higher arterial pressure tending to check blood stasis; and being deeply seated, it is better supported, and less exposed to injury.

The tibia is affected more often than any other bone.

In this disease the blood becomes charged with infective matter, not necessarily by contagion through a wound, for, as before said, all the signs of virulent pyæmia may arise without the local abscess having been opened. The theory of autogenetic origin is not borne out by facts, and this being granted, we are driven to suppose that the *materies morbi* is introduced by the skin, or, more likely, by the mucous



Fig. 42. — Total Necrosis of the Shaft of the Tibia, the result of acute Syphilitic Osteitis and Periostitis.

The sequestrum separated naturally, as shown by the irregular worm-eaten appearance of its ends. (One-third natural size.)

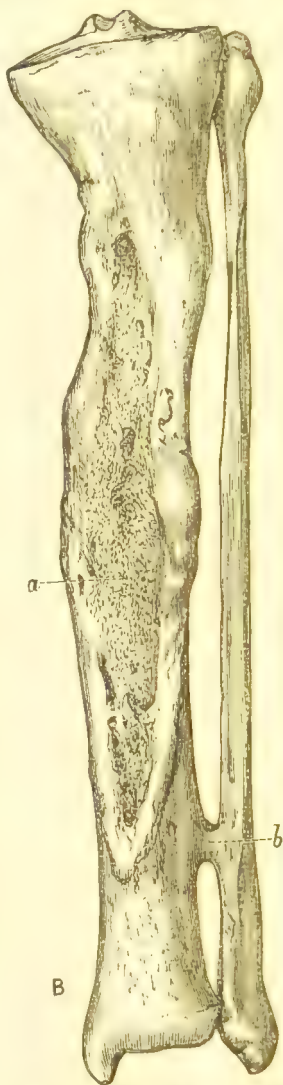


Fig. 43. — Showing complete Restoration of the Shaft of the Bone after original one had been destroyed by Necrosis. From the same subject as A.

a, Caries of the new bone; b, bridge of bone uniting the tibia with the fibula. (One-third natural size. Vide p. 268.)



membranes. The analogy furnished by the acute specific diseases supports this view. At the same time, there can be no doubt but that it finds a suitable nidus for development and reproduction in bone-tissue, the vitality of which has been lowered by injury or other cause.

The living structures strive to get rid of matter obnoxious to themselves, and do their best to destroy any organisms that infest the blood and tissues; but they may be overpowered by the intensity of the initial infection, or succumb to the continual absorption under high pressure from the seat of primary suppuration.

Early and free incisions, by lowering the tension, check the progress of the inflammation, and diminish the risk of blood-poisoning by providing for the escape of the exudation.

**Repair.**—The loss of the whole or greater portion of a shaft is replaced by new bone, deposited from any of the original periosteum that may have remained, and from the fibrous structures around; nor is it necessary, as was formerly supposed, that the necrosed bone should be left for a long time to act as a stimulus to ossification, for early “subperiosteal resections” are successful. I have seen complete restoration of the diaphysis of a tibia that was removed on the tenth day from the onset of the disease (Mr. W. Pye’s case).\* The younger the patient the greater is the developmental activity, and the more rapid the reconstruction.

If the epiphyseal cartilages have been destroyed, the new shaft will not attain to the length it otherwise would have done.

**Phosphorus necrosis of the jaws.**—The disease is less common than formerly; for red amorphous

\* *Lancet*, vol. ii. p. 654; 1879.



phosphorus is used instead of the yellow variety, and workers in phosphorus look more after the state of their teeth. It is caused by the acid fumes of phosphorus (probably phosphorous acid) acting upon exposed or unhealthy bone in the vicinity of *carious teeth*. The disease may begin as an acute osteo-periostitis, which rapidly ends in necrosis; or it may be preceded by an osteo-plastic inflammation. This depends on the intensity of the cause, and the extent of caries of the teeth and exposure of the alveolar process of the jaw. The final result is death of the whole, or a large portion, of the jaw. An exuberance of spongy vascular bone is deposited around the sequestrum, which often takes a long time to separate. Sequestrotomy has been known to cause severe bleeding; in one case the carotid artery had to be tied to arrest it.

**“Quiet necrosis.”**—Sir J. Paget has described a form of necrosis in which the death of the bone and separation of the sequestrum take place without manifest signs of inflammation. In fact, the patient may be quite ignorant of there being anything wrong. This “quiet necrosis” may be superficial or internal, or very rarely total. The sequestra lie in cavities lined by granulation tissue, but they excite so little irritation that no external openings are formed. The periosteum is “thickened, tough, and little vascular.” There is usually considerable thickening of the bone, and this, together with absence of pronounced signs of inflammation, has led to the mistaken diagnosis of sarcoma, and hence to amputation of the limb. A similar process is occasionally observed in connection with articular cartilage; slight injury appears to be the exciting cause.

## CHAPTER XXXIX.

## BONE ABSCESS.

BONE abscess is either the result of an injury, or constitutional disease, such as tubercle or syphilis. The favourite locality is the end of a long bone ; but it may be found wherever there is a wide tract of cancellous tissue, as in the tarsal bones and the vertebrae. Commencing in a rarefying osteitis, a cavity is formed by the gradual absorption of the bony trabeculae by granulation tissue ; this granulation tissue, in its turn degenerating, mingles its debris with exudation products, and the abscess is completed. At first its walls are composed of soft carious bone, which, if the inflammation spreads, gradually becomes disintegrated, the cavity enlarging at its expense.

But here the destructive process may end and an organising one begin. New bone is then deposited around the vessels of the granulation tissue, and this goes on until a zone of hard sclerosed bone has replaced that previously softened.

The layer of granulation tissue immediately lining the cavity is converted into fibrous tissue (a kind of endosteum) ; all formed elements found in the contents of the abscess break down from fatty degeneration, and only a serous or sero-purulent fluid remains. In some cases it would appear that the granulation tissue, having completely absorbed the bone for a considerable distance, does not liquefy, but undergoes caseation, the bone around becoming condensed, as described above.

More frequent than either of the above modes of termination is a progressive absorption of the bone, until the abscess opens by a narrow orifice,

either upon the surface or into the contiguous joint, or both.

At the outset the obstruction to the circulation in the bone may be so great that a portion becomes necrosed, and is afterwards set free in the cavity formed by the more gradual destruction (rarefying osteitis).

Bone abscess forms a conspicuous feature in the pathology of many cases of strumous disease of joints.

Care should be taken in probing them through a sinus opening externally, as they are often bounded next the joint by the articular cartilage alone, and this might be inadvertently detached.

When confined to the interior of the bone, and very chronic, the only symptom perhaps is localised aching, and the chief sign a fixed spot of tenderness to touch.

Once opened, they rarely close spontaneously, or, if so, only for a time. The tension being relieved, the sinus leading to the abscess shrinks until the re-accumulation of pus raises the pressure and again determines a discharge. Trephining, by giving a free exit, removes the tension which is the chief local cause of non-obliteration of these cavities. Granulation tissue then encroaches upon the space without hindrance, and organisation into fibrous tissue or bone puts an end to further trouble.

## CHAPTER XL.

## MOLLITIES OSSIUM—OSTEOMALACIA.

THIS disease is more common in females than in males. It affects chiefly the periods of early and mid-adult life ; it is not found in children. By some it is supposed to be a premature senile decay ; but atrophy of the bones of old people does not as a rule assume this form. We may safely conclude that its real cause is unknown.

Like rickets, it seems to be the expression of a general disease, or at least a morbid state of the osseous system, and not a mere local or accidental disturbance of nutrition in one bone or group of bones.

The greater part of the skeleton may be involved ; the vertebræ, ribs, pelvis, and long bones suffer most ; the bones of the cranium, carpus, and tarsus enjoy a much greater immunity.

**Morbid anatomy.**—In the long bones the disease commences in the central canal, and then extends to the medullary spaces and Haversian systems of the cancellous, and finally the compact bone.

The periosteum seems to take little or no part in the process ; in fact, Rindfleisch ascribes to it a conservative rôle, and says that, by nourishing the peripheral layers of compact bone, it checks, and even prevents, the outermost laminae from being absorbed. The disease is a progressive one, and rarely stops before the long bones are hollowed out into mere shells, encased in a parchment-like layer of bone, or riddled with cystic cavities ("cystic degeneration").

Billroth mentions two cases of local osteomalacia in the long bones of the extremity ; but, as both were

the subjects of caries of the joints, the osteoporosis was probably caused by rarefying osteitis and fatty atrophy from disuse; the microscopy is not given.

In the vertebræ, pelvis, sternum, and ribs it commences in several parts of the cancellous tissue at the same time, as shown by the numerous scattered excavations.

**Naked-eye appearances, etc.**—In the early stage there is very marked congestion of the medulla, in the central canals of the long bones, and the medullary spaces of the cancellous tissue generally. The dark-red colour seems to point to the congestion being passive rather than active. As the vessels are imperfectly supported, capillary ruptures take place. Later, cystic cavities are formed by absorption of bony trabeculæ and liquefaction of the medulla. These are filled with an albuminous fluid, clear, or turbid from fatty debris, and blood pigment the remains of extravasations. When the cysts cease to enlarge they become lined with a fibrous membrane, having but few vessels. This is derived from organisation of the outer portion of medullary tissue that originally filled the spaces, and from fibrous transformation of the decalcified bone. The attenuated osseous trabeculæ are so soft that they may be bent or cut quite easily.

Very scanty osteophytes are occasionally deposited beneath the periosteum (Billroth).

**Microscopy.**—The capillaries of the affected medulla in the central canals, cancellous spaces, and Haversian canals are greatly enlarged. They are embedded in a soft gelatinous substance composed of a homogeneous basis containing embryonic cells, some of which are filled with large fat-drops. The cells are much less numerous than in caries. There are, besides, pigment granules and free blood-corpuscles. The natural fat cells disappear.

The bony trabeculæ bounding the spaces are

somewhat festooned, but to nothing like the extent as one sees in the form of Howship's lacunæ in caries. The lime salts are absorbed before the animal constituents, so that there are two distinct zones around the medullary spaces, the one composed of soft decalcified bone, homogeneous from obliteration of the

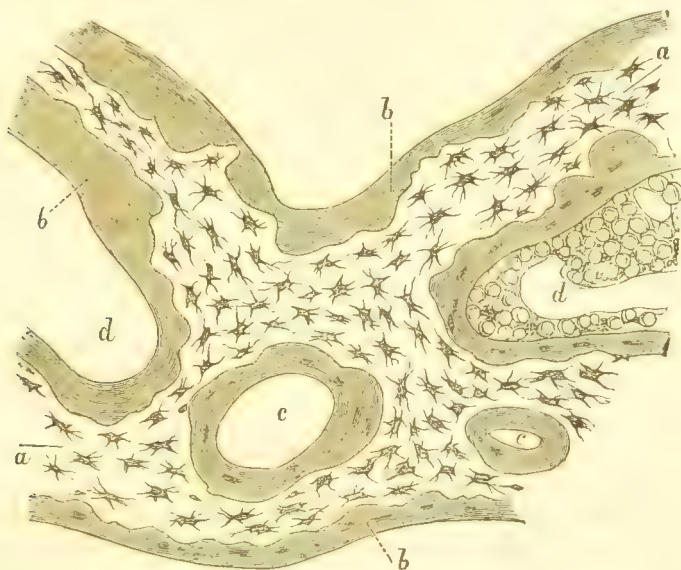


Fig. 44.—Mollities Ossium—Splinter of Bone from the Spongy Substance of an affected Rib.

*a*, Normal bone tissue ; *b*, decalcified bone tissue ; *c*, Haversian canal ; *d*, medullary spaces, the one on the left filled with red marrow. The capillary vessels are gaping widely. 1-300th. (After Rindfleisch.)

lacunæ and canaliculi, the other of healthy osseous laminae (Fig. 44). Billroth defines the disease as a "fungous fatty osteomyelitis."

**Chemistry of the disease.**—Rindfleisch suggests that the actual cause of absorption of the calcareous salts may be *free carbonic acid* contained in the blood of the congested medulla. This seems very doubtful. Weber has demonstrated the presence of



*lactic acid* in the urine and bones, but this is more likely the consequence than the cause of osteomalacia. There is an excess of earthy salts in the urine.

**Deformities.**—As the morbid process causes rarefaction and softening, the bones are rendered liable to curvatures and partial and complete fracture, the sum total of which is the widespread deformities of the skeleton. The mere weight of the superincumbent parts and the natural muscular contractions are sufficient to produce the most striking results; thus the ribs are bent by the pressure of the arm in the axillary line. They fall in near their sternal ends and along the attachment of the diaphragm. The weight of the trunk increases the natural curves of the spine, and the vertebræ at the same time undergo more or less rotation. The pelvis assumes a trefoil shape under the action of two forces: the weight of the body, which thrusts the sacral promontory forwards and downwards, and the counter-pressure through the acetabula.

The long bones may be the seat of many fractures, partial and complete.

**Mollities ossium contrasted with caries.**—

(1) Mollities ossium attacks many bones at the same time; (2) each bone is the seat of scattered foci of absorption and excavation; (3) the lime salts are absorbed before the organic constituents; in caries there is simultaneous removal; (4) the neoplasia softens and liquefies, but does not caseate; (5) the cavities formed in the bones have regular walls, which are sometimes lined with a fibrous membrane; (6) the disease has but little tendency to subside; and (7) it does not begin nor spread beneath the periosteum.

## CHAPTER XLI.

## DISEASES OF THE JOINTS.

WHEN studying the pathological changes going on in an inflamed joint, it is well to direct a systematic attention to the character of the secretion, the synovial membrane, articular cartilage and bones, the fibrous capsule, and interarticular ligaments where these exist. It will be found that the anatomical components of a joint are modified individually or collectively, in different degrees and in varying manner, according to the nature of the cause—*e.g.* :

*Simple traumatic arthritis* is characterised by the large amount of serous exudation, with but little tendency to the formation of pus, whilst the cartilage and bone are practically untouched.

*Tubercular arthritis* commonly ends in suppuration and “ulceration of cartilage and bone.”

*Gouty arthritis* is marked by a slow but permanent change in the articular cartilage, deposit of crystals of urate of soda, and recurrent painful attacks of effusion.

*Pyæmic arthritis* causes a marvellously rapid purulent effusion from the synovial membrane, often with comparative freedom from pain.

In *chronic rheumatic arthritis* the most noteworthy features, from a clinical point of view, are the extreme chronicity and steady downward course, from an anatomico-pathological, the scantiness of the exudation (dry arthritis), the absorption of the articular cartilages, the eburnation of the bones, and the exuberant growth of osteophytes.

*Gonorrhœal rheumatism* is often very intractable, and liable to recur again and again.

**Ætiology.**—The causes of arthritis may be classified as follows:—

1. *Traumatism*, including the cases that arise from the spreading of inflammation in the continuity of tissue from other parts—*e.g.* a diseased bone.

2. *Acute blood-poisoning*: (*a*) the acute specific fevers; and (*b*) pyæmia, syphilis, gonorrhœa.

3. *Diathetic states*: gout, rheumatism, hæmophilia.

4. *Trophic lesions* and vaso-motor paralyses: loco-motor ataxia.

5. *Degeneration from old age*, which may underlie simple senile arthritis, and predispose to tubercular inflammation; “senile scrofula.”

Attention to the previous state of nutrition of the articular structures must not be omitted. When we say that a certain poison selects a certain tissue for its local manifestation, the truth is only half expressed. The tissues play a very important part in the history of the causation of their morbid states. This is nowhere better exemplified than in the joints. In gout there is a great predilection for the tarso-metatarsal articulation of the great toe. In scrofula it is the hip, knee, and the joints of the hands and feet; in dry arthritis the hip, shoulder, knee, and temporo-maxillary articulation, and the digits; in gonorrhœal rheumatism the knee and wrist. Previous disease, whilst it increases the liability to subsequent attacks, seems to confer a quantitative protection in some cases. Thus, if a healthy joint be wounded, there is great danger of acute destructive inflammation, general constitutional disturbance, and high fever. It is very different in the case of one damaged by chronic strumous arthritis. Parallel cases are furnished by the normal peritoneum and an old thickened hernial sac.

## CHAPTER XLII.

## TUBERCULAR ARTHRITIS.

**Synonyms.**—White swelling; fungous disease or pulpy degeneration of the synovial membrane; ulceration of cartilage.

**Causes.**—Hereditary or acquired weakness of the tissues may act as a predisposing cause. An injury may determine the onset and site of the inflammation, but the agent which stamps its pathological identity is the tubercle bacillus.

**Morbid anatomy.**—The disease begins in the synovial membrane or the bone, never in the cartilage. Pathologists differ as to the relative degree of frequency of the two sources. Many believe that in a great majority of cases it starts in the synovial membrane. My own observations lead me to conclude that in not a few instances the bone is primarily affected, especially in the carpus and tarsus. In the hip-joint the ligamentum teres is sometimes held to be the seat of the initial lesion, but then it is really the synovial membrane that surrounds the ligament.

At first the *synovial membrane* is of a bright pink colour; it looks glistening, and is slightly swollen. The microscope shows dilatation of the capillaries, and some leucocytes. Later, the swelling becomes more marked; the surface endothelium is shed, and the meshwork of areolar tissue is no longer visible. Its fibres have softened and melted away, the whole being overrun with wandering cells. In short, the folds and fringes have been converted into a reddish-grey mass of gelatinous granulation tissue. Miliary tubercles can sometimes be demonstrated in the synovial membrane.

By this time the *cartilage* is involved at the

periphery ; it has lost its pearly lustre and firm consistence. The change consists of a mucoid liquefaction of the matrix and a proliferation of the cells. Vertical sections show successively from the free surface a layer of indifferent cells, broods of cells in process of joining one another, cartilage corpuscles in various stages of segmentation, and healthy cartilage.

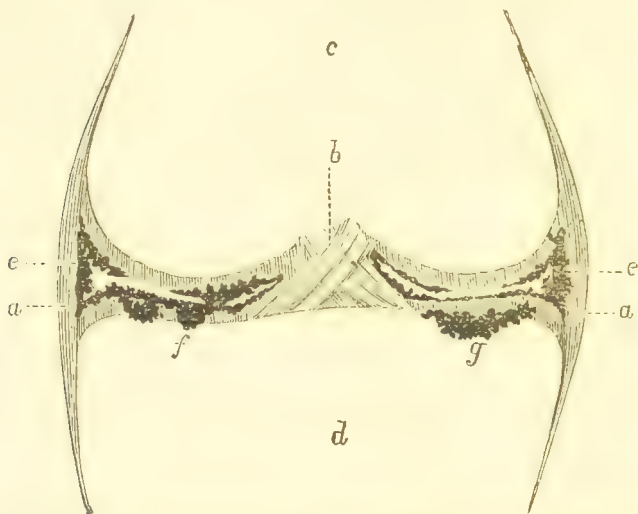


Fig. 45.—Diagram of a Section of a Knee-joint (the Interarticular Cartilages have been left out, the Articular Cartilages shaded), with Fungous Inflammation.

*a, a*, Fibrous capsule ; *b*, crucial ligament ; *c*, femur ; *d*, tibia ; *e, e'*, fungous synovial membrane growing into the cartilage ; at *f* it even grows into the bone ; at *g* isolated proliferations of the granulations into the bone on the border between bone and cartilage. (After Billroth.)

The *fluid* in the joint is now thin and cloudy from the presence of leucocytes and fat particles.

The fungous granulation tissue goes on increasing by the development of new vessels and continued exudation until it fills all the crevices of the joint, pushing its way between the bones, and rooting its vessels in the softened cartilage as it passes over it. At length the entire thickness of cartilage is destroyed

in patches, and the bone is attacked with rarefying osteitis (caries).

In some cases the cartilage is absorbed on the articular and osseous surfaces at the same time, so that flakes are set free in the joint, whilst other portions are so loosened from their attachment to the bone that the handle of a scalpel can be readily thrust between the two structures (Fig. 45). The earlier pathologists, struck by this state of things, named the disease "ulceration of cartilage"; but, as before said, this is always secondary to fungous synovitis or osteitis.

"Starting" pains indicate not only that the cartilage is ulcerated, but that the bones are exposed in the joint.

By this time the joint usually contains pus, and molecular and shreddy debris of broken-down granulation tissue and cartilage. Instead of diffuse suppuration, localised abscesses may form in succession in different parts of the joint, open externally, discharge for a while, and then close, leaving the permanent cicatrices in the soft parts, so often seen about partially dislocated, stiff, or ankylosed joints.

When the bones are diseased, either primarily or as the result of extension of inflammation from the joint, they are affected with tubercular rarefying osteitis (pages 259-261).

*The capsule* is softened and thickened. On section it looks gelatinous. It is stretched by the pressure from within, except on the aspect of flexion, where it is more or less contracted.

*The tissues around* suffer considerably.

*The muscles* waste from disuse. They become fatty, and undergo interstitial absorption; hence the shortening.

*The subcutaneous tissue* is congested and oedematous. Except in very acute cases, *the skin* retains its



white appearance (tumor albus). The *ends of the bones* appear enlarged, but this is misleading. It is mainly due to swelling of the soft parts and the shrinking of the muscles.

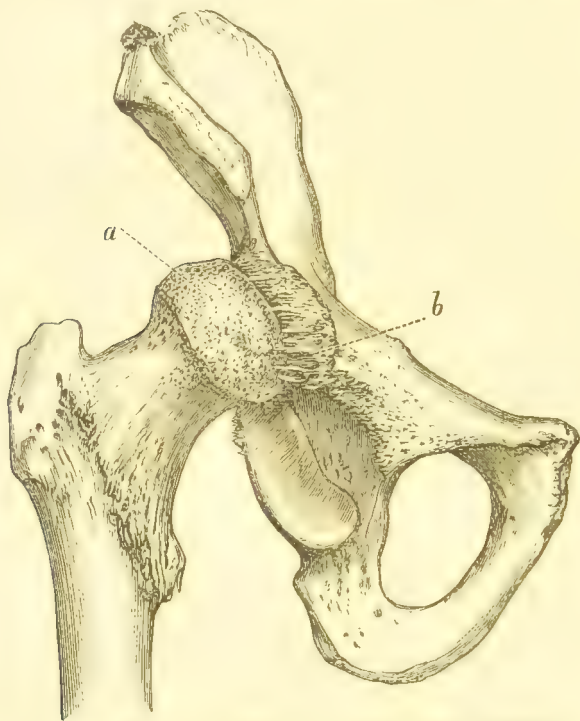


Fig. 46.—Hip-joint affected with Tubercular Arthritis.

The head of the femur, *a*, has lost its cartilage; the articular surface is composed of porous, rarefied, cancellous tissue; the upper and posterior part of the acetabular rim has been absorbed by the joint action of the carious process and the continuous pressure of the head of the femur; *b*, buttress of bone, composed of sharp stalactitic osteophytes thrown out to support the dislocated head. (One-third natural size.)

Abscesses not unfrequently form outside the articular capsule.

**Flexion and dislocation of the joint—*e.g.*** the knee. Flexion is caused by reflex contraction of the muscles (Hilton), and by the hydrostatic pressure

of the effusion within the joint (Bonnet). The head of the tibia is rotated out and displaced backwards and outwards. Several forces concur in effecting this—atrophic contraction of the muscles, shrinking of the capsule and external ligaments, destruction of the crucial ligaments, and the weight of the limb.

**Terminations of the disease.**—It may be arrested at any stage; perfect movement may be restored, more often stiffness remains. Anchylosis is not uncommon. In this case the granulation tissue between the bones organises to fibrous tissue or bone: to fibrous tissue only if the cartilage is not destroyed in its entire thickness. Suppuration and complete destruction of the joint with extensive disease of the bones are very common.

**Hip-joint disease.**—In some works the minute anatomy of hip-joint disease is given at great length, as though it constituted a special disease. This is not the case. It is tubercular arthritis (white swelling), and begins, as before said, either in the synovial membrane, or the bone, as implied in the enumeration of the “articular,” “femoral,” and “acetabular” varieties. What makes the affection so important is its frequency, anatomical complexity, and clinical gravity.

The abscesses that form in connection with the disease point above and behind the great trochanter, or in the groin, following the course from the joint taken by the internal circumflex artery.

The psoas bursa may be involved, and this result is more likely to happen when there is a natural communication between bursa and joint.

When the acetabulum is extensively diseased, pus often collects within the pelvis, and the head of the femur, or what remains of it, is sometimes forced through the perforated floor of the cavity by the functional and atrophic contraction of the muscles, or by accidental pressure.

## CHAPTER XLIII.

## CHRONIC RHEUMATIC ARTHRITIS.

**Synonyms.**—Arthritis deformans, proliferating arthritis, dry arthritis, nodular rheumatism, *malum coxæ senilis*, rheumatic gout, osteo-arthritis. These terms refer to some points in the clinical or pathological history of the disease, which, although it presents wide structural variations, is well defined in its general features and anatomical lesions.

**Causes.**—The ætiology is obscure as regards the immediate cause of the particular tissue change. It is met with chiefly in persons beyond mid-life, and in those exposed to inclemencies of climate.

The disease occurs under two forms, according as it is localised in one or more joints.

In the former case it attacks the large joints, especially the hip, and as it is incidental to advanced age it has been called *morbus coxæ senilis*. It is mild from the first, and is rarely traceable to injury.

The polyarticular variety is found in the small and medium-sized joints, those of the digits in particular. It affects the young and middle-aged rather than the old; women more often than men. It begins as a distinct affection, or it is the sequel of acute articular rheumatism.

**Morbid anatomy.**—In the majority of cases the cartilages are first affected, but the synovial membrane, bones, and ligaments soon become involved. It never ends in suppuration. In this way it stands in marked contrast to strumous arthritis.

The disease is characterised by (1) a proliferation and subsequent destruction of the articular cartilage; (2) by eburnation of the ends of the bones; (3) by

the formation of ecchondroses and massive rounded osteophytes; (4) by chronic inflammatory hypertrophy of the synovial membrane.

The joints of the fingers and, less frequently, the toes become knobbed, stiff, and contracted (*rheumatismus nodosus*).

The internodal portions of the phalanges waste very much from disuse in the later stages of the disease; in fact, they may almost entirely disappear.

**Changes in the cartilage.**—These are essentially proliferating or constructive; but, inasmuch as the consistence is diminished, the new-formed cells and softened matrix are gradually worn away and finally destroyed, leaving the bone exposed and condensed.

The surface layer of cells is first affected. The cells multiply by segmentation, but so slowly that instead of forming small round unstable granulation corpuscles, as is the case in white swelling, they assume more or less the characteristics of cartilage elements.

The primary capsules enlarge and fill with numerous secondary capsules. The secondary capsules are contained one within another, forming a series of concentric rings, or they remain isolated within the primary capsules, or, if this has disappeared, in one large capsule.

By softening of the matrix, the cavities filled with new capsules and cells open into one another, constituting alveolar spaces perpendicular to the articular surface, very similar to what takes place at the border of normal ossification of bone, and in the epiphyseal cartilages in rickets. The superficial capsules dissolve and set their contents free into the joint, leaving soft, wavy, villous processes of the matrix.

The more or less parallel and vertical arrangement of the cell groups and bands of matrix is due

(1) to the lateral resistance to expansion of the enlarging capsules; (2) to the normal construction of cartilage, which, although it appears homogeneous on section, is seen on fracture to consist of columns set at right angles to the bone—*i.e.* in the direction of greatest pressure. At length the bone is exposed.

**Changes in the subcartilaginous bone.—**

This also undergoes inflammatory absorption, but as the vascularisation and initial rarefaction are outstripped by the condensation immediately below the surface, the spongy cancellous tissue is not exposed. The sclerosing osteitis causes eburnation. The bone is gradually worn away and polished by friction, either uniformly or in furrows.

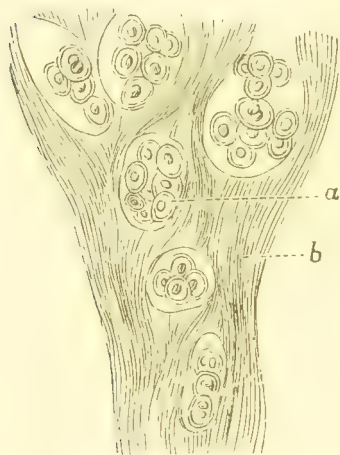


Fig. 47. — Nodular Rheumatism.  
Surface of the Cartilage.

*a*, Primary capsule filled with secondary capsules about to open into the articulation; *b*, segmented matrix. Magnified 200 diameters. (After Cornil and Ranvier.)

**Changes in the synovial membrane.—**The synovial membrane becomes more vascular, and swollen and thickened. The villi or fringes stand out as club-shaped protrusions. New processes

are given off from the old ones, so that a branched appearance is produced (“arborescent budding”).

The fat cells disappear, and their place is taken by exudation corpuscles, some of which escape into the joint, and render the fluid, now increased in quantity, more or less turbid. But the greater part of the inflammatory neoplasia organises into dense connective tissue. Some of the indifferent corpuscles develop in cartilage, so that nodules are formed, and

these remain isolated, or coalesce into thick tuberculated masses (Fig. 47).

Meanwhile, the lateral portions of the articular cartilages, less subject to pressure and friction than the central part, whilst they proliferate, are not worn away, but, being soft, they yield like indiarubber to a force insufficient to cause their destruction. Thus the articular surface of the bone is greatly widened. In the case of the hip-joint the cartilage (subsequently bone) droops, like a mantle with wavy border, from the head of the femur (Fig. 48).

The cartilaginous growths in the synovial membrane sometimes form pedunculated masses. They frequently calcify, and ossify into spongy bone. They are liable to be detached and roll free in the joint as foreign bodies. They sometimes appear as though "glued on to the bone."

The **secretion in the joint** is thin, and clear or cloudy, never purulent. In some instances it is considerably increased, especially in the earlier stages of the inflammation. In others there is scarcely any at all; this is notably the case in the polyarticular variety, which affects the digits (dry arthritis).

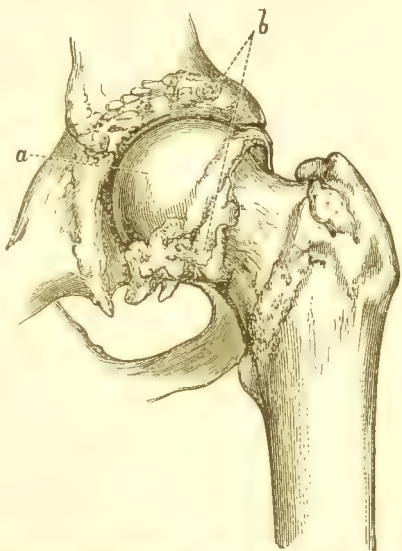


Fig. 48.—Hip-joint affected with Chronic Rheumatic Arthritis.

*a*, Articular surface of the femur denuded of its cartilage, but smooth from the eburation consequent on sclerosing osteitis; *b*, rounded osteophytes. (Compare this figure with Fig. 16. (One-third natural size.)



The **extra-articular structures**.—The muscles of the limb waste from disuse. The ligaments become dense and contracted, frequently ossified; so also the tendons attached to the bones around the affected joint.

Osteophytes spring up from the periosteum, and grow into the articular capsule. Many are isolated, and lie at some little distance from the joint.

The absorption of the head and shortening of the neck of the bone, together with the extensive out-growths, give to the hip-joint *e.g.* a remarkable appearance, justifying the name, *arthritis deformans*.

## CHAPTER XLIV.

## ACUTE SEROUS SYNOVITIS —HYDROPS ACUTUS.

TAKE a knee-joint that has been injured by a blow or strain, or that has inflamed from exposure to wet and cold ; it soon becomes painful, especially on movement, so that walking is difficult. The pain is of an aching character, from stretching of the nerves. Swelling comes on very quickly, and so rapid is the effusion that the lateral depressions by the patella and patellar ligament, and the pouch beneath the triceps muscle, may be raised into prominences in a few hours.

The skin over the joint retains more or less its natural colour. The joint is somewhat flexed, but not to the extent that is found in acute suppurative arthritis, although the amount of fluid it contains may be much greater than in the more severe affection. This seems to prove conclusively that the chief cause of the flexion lies in the reflex contraction of the muscles set up by the irritation of the nerves supplied to the synovial membrane.

If the fluid be drawn off by the aspirator it will be found to be clear, or slightly cloudy, from shedding of the synovial endothelium, exudation of leucocytes, and it may be formation of fine flakes of fibrin, and strings of mucin.

If such a joint be opened the synovial membrane will be found to be uniformly pink, puffy and gelatinous, showing no signs of suppuration or disintegration. Under the microscope the fibrous element looks glassy, and is seen to contain exudation cells.

When the inflammation is unusually severe, or resolution long delayed, the articular cartilage becomes

cloudy on the surface, perhaps a little softened. The superficial cells are granular and swollen, and sometimes in process of segmentation. The primary cartilage capsules are enlarged; the secondary ones more numerous. The matrix is somewhat fibrillated.

This form of synovitis resembles inflammation of the large serous membranes (pleuræ, pericardium) in the rapidity with which the synovial membrane pours out large quantities of fluid.

Unless the articular lesion is the result of acute rheumatism, the constitutional disturbance is not great.

As the inflammation resolves the pain goes, and the function of the joint is restored. Frequently, however, it becomes chronic, the fluid remaining in excess.

## CHAPTER XLV.

## CHRONIC SEROUS SYNOVITIS.

THIS disease, called also hydrops articuli, is either the sequel of acute or subacute synovitis, or it is obscure in its mode of origin. It is generally met with in the knee-joint, and it affects by preference young adult males. The signs of inflammation (save one, swelling) are so little marked that some pathologists regard the inflammation as a simple dropsy, analogous to hydrocele of the tunica vaginalis testis; but from the fact that it is often the remains of a more acute process, and that in a series of cases every gradation may be met with, from a slow painless effusion to a fairly rapid exudation, accompanied by increased heat of the joint, this view seems untenable.

**Morbid anatomy.**—The *fluid* may be slightly viscid, but as a rule it is thin and serous. The *synovial membrane* is slightly swollen and cedematous. In old cases it is somewhat indurated from the growth of a low form of connective tissue, but it never presents the gelatinous fungous appearance of white swelling, and the tendency to suppurate is almost nil. The capsule is stretched, but the joint, though crippled mechanically, is very little, if at all, painful. The spontaneous form of the disease is often symmetrical. This points to a local inherent weakness of tissue, or to some constitutional disorder. Some cases have been shown to be tubercular. The greater the viscosity or colloidal nature of the fluid, the more difficult is its absorption.

## CHAPTER XLVI.

## ACUTE SUPPURATIVE ARTHRITIS.

**Causes:** (1) Severe injury, especially wound of the joint; (2) spreading of acute inflammation from the bones or soft parts; (3) some infective material in the blood, that of pyæmia, or, more rarely, acute rheumatism, or the specific fevers.

**Pyæmic suppurative arthritis** sets in very rapidly; the joint may be distended with pus in a few hours. In these cases the synovial membrane is injected and swollen, but not always to the extent that one would expect from the amount of exudation. The cells are mainly derived from the vessels of the synovial membrane. Segmentation of the cells of the articular cartilage furnishes others when this structure is softened and eroded. There may be embolic infarctions and localised disintegrations of the synovial membrane; but this is the exception.

**Traumatic suppurative arthritis.** -Here the joint is intensely painful, sharply flexed, and immovable from spastic contraction of the muscles. The outline is somewhat globular; for the capsule, subcutaneous cellular tissue, and skin are all injected and inflamed; hence the elevations caused by the distension of the synovial pouches are lost in the general swelling.

The *synovial membrane* is intensely red, puffed, and pulpy. It may contain minute yellowish foci of interstitial suppuration. The vessels are dilated and pouched; in some stasis has occurred. The surface has lost its polish.

The *cartilage*.—This is obviously affected; the superficial portion is softened, pulpy, and either

granular or slightly villous. The microscope shows an active proliferation of the cells, and a homogeneous or fibrillar appearance of the matrix. The secondary capsules are dissolved, and the primary ones filled with embryonic cells. Next to the joint no trace can be seen of the original structure; nothing but a layer of indifferent cells embedded in the liquefied matrix.

The inflammation of the cartilage is secondary to that of the synovial membrane, and is set up by spreading in the continuity, and by the irritation of the exudation.

The *articular capsule* is thick and gelatinous, and the surrounding cellular tissue congested and œdematous.

The *contents* of the joint consist at first of an increased synovial secretion. Very soon the cavity is filled with pus.

If the inflammation is allowed to go on unchecked, the cartilages are entirely destroyed and the bones exposed; the capsule softens, and is converted into granulation tissue; the suppuration is no longer confined to the joint, but extends rapidly beneath the fascia around, or an external opening is formed, and so the tension is relieved.

There is severe constitutional disturbance, with high fever, the result of absorption of infective material, and the acute pain.



## CHAPTER XLVII.

GONORRHOICAL ARTHRITIS—GONORRHOICAL RHEUMATISM—  
URETHRAL RHEUMATISM.

GONORRHOICAL rheumatism is generally considered to be a mild form of pyæmia, due to absorption from the inflamed urethra. Bumstead and Taylor, however, reject this theory. Many eminent authorities hold that gonorrhœa itself is not a specific disease, but a simple acute suppurative urethritis; but even if it be conceded that it is due to a specific micrococcus causing a local infective inflammation, there is certainly not sufficient evidence to show that gonorrhœal arthritis is dependent on general infection with such organisms.

It occurs in only a fractional percentage of persons affected with gonorrhœa, and must therefore be supposed to result from some alteration in the composition of the discharge; or, what is far more probable, from a disposition of the synovial membrane in certain individuals to become inflamed on slight irritation. Painful swelling of the joints is far from rare in many inflammatory diseases, particularly those attended with suppuration. The simple passage of a catheter has been known to produce the same result. The disease is sometimes very obstinate, and is liable to recur long after the original cause has disappeared, and is almost certain to do so if it be renewed.

It may be accompanied by inflammation of the sclerotic coat of the eye-ball.

The pathological changes are the same as in acute and subacute synovitis arising from other sources.

The knee-joint is more often affected than any other. The inflammation generally subsides after a time, but it may go on to complete destruction of the articulation.

## CHAPTER XLVIII.

## HÆMORRHAGE INTO JOINTS.

CAUSES: (1) Injury; (2) rupture of a popliteal aneurism into the knee-joint; (3) hæmophilia; (4) some blood diseases—*e.g.* scurvy.

**Traumatic hæmarthrosis.** — This follows blows on the joint. The extravasation takes place from the capillaries of the synovial membrane, and, in the knee-joint, from the broken surfaces of a fractured patella. It is diagnosed from serous effusion by the fluctuation being less marked, and the rapidity with which the swelling follows the injury.

As a rule, the blood is entirely absorbed, but occasionally fibrinous coagula remain.

**Hæmorrhage from hæmophilia** is most common in the knee-joint. It is followed by a certain amount of synovitis.

Hæmophilia is eleven times as frequent in males as in females, though the latter usually transmit the disease (W. Legg). Beyond its consequences, the morbid anatomy is unknown.

## CHAPTER XLIX.

## LOOSE OR MOVABLE BODIES IN JOINTS. CYSTS CONNECTED WITH JOINTS.

THESE movable bodies are of different kinds :

(1) The most common are termed *melon-seed bodies*, on account of their prevailing shape, rounded at one end, somewhat acuminate at the other, and flattened. Some are as small as a hemp seed ; others are many times as large.

A healthy synovial membrane is finely fimbriated ; the primary fringes consist of delicate areolar tissue, covered by a layer of endothelial cells ; and loops of capillary vessels. The secondary fringes are sometimes extravascular (Rainey). Kölliker has shown that the synovial processes occasionally contain a few cartilage cells.

Whether from chronic inflammation or a pure hypertrophy, a part or the whole of the synovial membrane of a joint may be covered with small pedunculated bodies. In cases primarily inflammatory, and notably in those resulting from injury, the enlargement of the fringes is generally distributed over a limited area.

The pedicles are often extremely fine, and the least force suffices to rupture them. The melon-seed bodies, then, are clearly enlarged synovial fringes. Their microscopical structure varies ; in most cases they show nothing but a dense homogeneous fibrinous material ; sometimes there is a faint concentric lamination ; very rarely cartilage cells can be seen.

(2) Rounded or nodular masses of a gelatinous substance, devoid of any trace of organised structure. They are either fibrinous exudations from the synovial

membrane, or swollen degenerated synovial fringes (1 and 2 are also found in synovial sheaths, ganglia, and bursæ).

(3) Bodies of much larger size than any of the

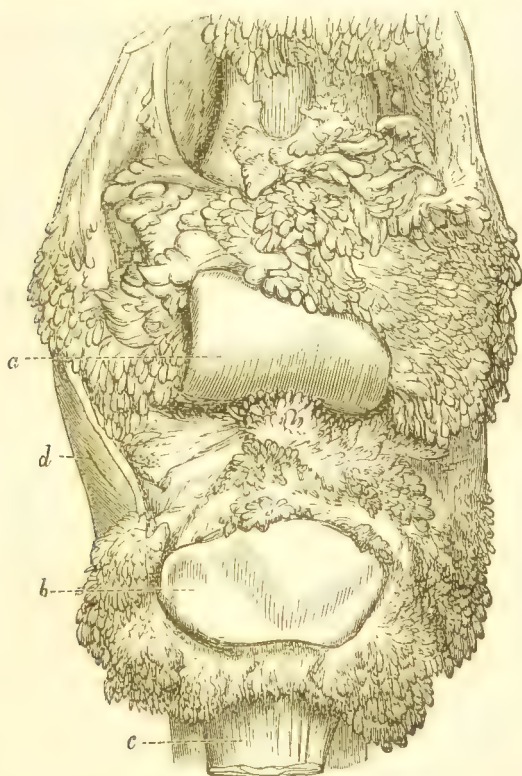


Fig. 49.—Lipomatous Hypertrophy of the Synovial Fringes of the Knee-Joint.

*a*, Articular surface of the femur ; *b*, patella ; *c*, ligamentum patellæ ; *d*, portion of capsule.

foregoing, consisting of hyaline or fibro-cartilage, either in the natural state, or ossified or calcified. They are arborescent outgrowths of the synovial membrane, or nodular thickenings of the lateral

portions of the articular cartilages in chronic rheumatic arthritis.

(4) Lipomatous bodies, hypertrophies of the cellulo-adipose tissue of the synovial membrane (Fig. 49).

(5) Portions of articular cartilage split off by accident, or set free by ulceration (white swelling).

(6) The remains of blood-clots.

(7) Synovial cysts.

Each of these groups is chiefly met with in the knee-joint.

**Morrant Baker's cysts**, or protrusion of pouches of synovial membrane through the capsule of the joint, commonly occur, but they are also met with in other joint affections—tubercular synovitis. The main swelling may be at a considerable distance from the articulation—*i.e.* in the calf of the leg, a narrow channel extending between the two.

## CHAPTER L.

## JOINTS AFTER INJURY AND DISEASE.

WITH regard to the morbid anatomy of arthritis, attention has so far been chiefly drawn to the changes observed during the progress of the disease. A few lines may be added explanatory of the permanent damage left after the pathological storm has passed by. The signs of "old joint disease" are very varied, but they will be quite easily understood if the chief factors in determining the result are borne in mind. In the first place the *cause* of the inflammation and its degree of severity must be kept prominently forwards; other things being equal, there will be greater destruction of tissue and increased liability to general fibrous or osseous ankylosis after strumous than simple traumatic arthritis, provided always that the processes of repair are not altogether prevented. The *amount of serous effusion* has a direct bearing on the shape and arrangement taken by the other inflammatory products—*e.g.* in chronic synovitis from injury to a healthy joint. The fluid may be sufficient to float and at the same time keep asunder any enlarged synovial fringes, and so prevent their joining one another and forming fibrous *adhesions*. It must not be concluded, however, that because the fluid is in excess the synovial membrane must necessarily present an arborescent appearance, for in some cases of chronic hydrops there is very little proliferation of tissue.

The **extent of movement** in the joint is not without its influence; the more this is restricted the greater is the atrophy of the bones and surrounding soft tissues, and the more likelihood is there of complete fixity from ankylosis, for limitation of



movement gives physiological rest to the inflamed tissues, and so an opportunity for their organisation and repair—*e.g.* in white swelling. Again, movement means friction, and unless there be enough fluid to counteract its effects, the solid products of inflammation, which are ill fitted to resist pressure, get worn away; the extremely varied appearances of joints affected with chronic rheumatic arthritis (a disease essentially proliferative) is largely owing to the degree of movement; when this is very slight the opposed articular cartilages, instead of being eroded, and the bone eburnated and polished, remain covered as it were with an eruption, which looks like drops of tallow or wax.

Movement is one cause of elongation of the pedicles of enlarged synovial fringes.

The **state of the extra-articular structures** is worthy of notice. In the course of arthritis, the muscles, tendons, and ligaments are unequally affected by atrophy from disuse, and by inflammatory softening and subsequent cicatricial shortening. These conditions, aided by the other forces of displacement (fluid effusion and physiological contraction of muscles), alter the natural lines of pressure, and so play an important part in fashioning acquired articular deformities.

**Changes in the articular cartilage.**— In *strumous arthritis* the articular cartilages are liable to marked variation according to the extent and distribution of the morbid process; in one case they may escape erosion except at their peripheral portions; in another the surface layer is converted into granulation tissue, which may organise, with or without fibrous ankylosis of the joint. In the latter event a seam of fibrous tissue is bounded on one or both sides by the remains of the cartilage; or, lastly, the whole of the cartilage may be destroyed, and that, too,

without the occurrence of suppuration. The author has seen the femur, tibia, and patella fused into one mass of compact bone, which, except for the absence of an operation scar, might easily have been mistaken for an old excision. One sometimes comes across a preparation in which there is a localised loss of substance in the articular cartilage, and the question arises, Was it of traumatic or pathological origin? If the loss of substance is the only abnormal sign, or if the adjacent synovial fringes are in a state of simple hypertrophy, it is traumatic. On the other hand, if the cartilage is undermined, the subjacent bone eroded, and the synovial membrane pulpy, it is evidently a specimen of tubercular disease. Furthermore, when tubercular arthritis begins in the bone the cartilage is usually first destroyed at or near the centre of the articular surface.

The state of the cartilage in *chronic rheumatic arthritis* has been fully described.

In *gout* the cartilage may be rendered so brittle by the deposit of urate of soda in its substance that plates may be chipped off or portions worn away by the movements of the joint or by injury.

## CHAPTER LI.

## ON DEFORMITIES.

AMONGST the deformities of greatest interest to the surgeon are those involving: (1) Malposition of the bones entering into the articulations of the extremities; and (2) curvatures of the spine.

The shape of the bones may remain unaltered; or it may deviate widely from the normal, as a part of the initial lesion, or as a secondary result of misdirected and excessive pressure.

**Causes.**—The causes of deformities are numerous:

(A) *Arrested or perverted development* during intra-uterine life. Such are the congenital defects in the different varieties of club-foot, dislocation of the hip, spina bifida, and meningocele (cerebral). That most of the forms of club-foot are due to abnormal foetal evolution is shown by the great regularity of disposition of the structures implicated; thus we come to arrange them in certain well-defined groups. Again, the most common (talipes varus) is mainly an exaggeration of the position assumed during complete rest—i.e. when all voluntary muscular action is withdrawn, as in the cadaver. Dieffenbach says “that all children are born with the first stage of club-foot.” This is only true in a physiological sense, for when the child comes to walk the foot takes its natural position without let or hindrance from alteration in the form of the articular surfaces of the bones, or contraction of ligaments, or paralysis or shortening of muscles. In early foetal life the back of the lower extremity lies in contact with the belly, and in very rare cases it has been known to be fixed there by integument common to it and the trunk. As

development goes on, *the limb rotates*, so that the calf comes to look backwards, and the sole of the foot downwards. Talipes varus may be considered as the result of imperfect rotation. It is not uncommon for spina bifida to be associated with some variety of club-foot, and pathologists have sought to connect the two by supposing that the latter is due to perverted innervation. But club-foot usually occurs without any other sign of disease of the spinal centres or nerves. In one case I found the brain and cord absent, adhesion of certain fingers to the palm, extroversion of the bladder, and *talipes calcaneus*. It seems far more probable that the spinal and pedal deformities are mere coincidences than that they stand in relation of cause and effect—at any rate, during intra-uterine life. The only structural changes are those due to arrested development and growth; there is nothing of an inflammatory nature.

*Intra-uterine pressure* explains certain irregular defects, and probably those of constant and definite type.

Cruveilhier held that talipes varus was the result of pressure combined with original malposition, the latter condition being probably due to a preliminary defective development.

The researches of R. W. Parker and Shattock led these observers to draw pretty much the same conclusion as regards the effect of pressure. In support of this theory they adduce the following facts:—

(1) Congenital subcutaneous bursæ, covered by patches of atrophied skin, have now and again been found over the prominent part of the foot.

(2) The tarsal bones are much altered in shape—*e.g.* the anterior part of the os calcis is incurved in congenital varus, so that the plane of the cuboid facet is directed unnaturally inwards; and the navicular facet of the astragalus is subdivided into two parts, which meet at varying angles, the inner portion

of the facet being applied to the displaced navicular bone, and the outer left unoccupied in consequence of the displacement. In one instance bursæ developed between it and the skin.

It is a point of morphological interest that in the anthropoid apes the outer border of the navicular facet of the astragalus is well inside the antero-posterior line which runs through the middle of the trochlear surface of the same bone and parallel to its inner border. In the normal human fœtus at birth this line touches the outer border of the facet; in the adult it intersects the outer part of the articular surface. The obliquity of the astragalus in the ourang gives an angle of  $45^{\circ}$ , which was found exceeded by  $5^{\circ}$  in a case of talipes varus.

(3) There is sometimes a malposition of other bones than those of the feet. Shattock records an instance where the fibula and tibia were so twisted on their long axis that at the lower end the former came to lie in front of the latter.

(4) The relative amount of liquor amnii varies at different periods of pregnancy; thus, after the fourth or fifth month the proportion is so small that the uterine environment of the fœtus begins to tell on its position. From the fifth month onwards the natural direction of the feet is towards the aspect of flexion (Lusk). It may be stated generally that the first normal position of the foot is that of varus, and the second—*i.e.* after the fifth month—calcaneus. If these physiological constants become exaggerated, we have the corresponding deformities. Hueter pointed out the fact that in no case can the thigh and leg be quite straightened at the time of birth.

(5) In some preparations there is evidence of want of movement in the ankle-joint in the shape of well-marked adhesions.

(6) Talipes varus is not unfrequently one-sided.

(b) *Paralysis and contraction of muscles.*—The theory of antagonistic contractions holds good in but a limited number of cases. (We refer to the physiological contraction, and not the atrophic shrinking from forced rest in unnatural position.) After *disease of the hip-joint*, especially if complicated with dislocation on to the dorsum ilii, the heel is drawn up (talipes equinus) to *compensate* for the shortening of the limb and tilting of the pelvis. In this instance there is both dynamic and static contraction of the calf muscles.

In *infantile paralysis* certain groups of muscles often remain permanently paralysed or weak, but the opponents do not drag the foot in the opposite direction; the position assumed is that caused by the weight of the foot, and any accidental pressure that may bear upon it.

Both in this and the congenital forms of club-foot the deformity is afterwards increased by misdirected pressure, as when the patient comes to walk upon the outer part of the dorsum in the latter case, or drags the limb after him in the former.

The position taken by the hand and fore-arm in injury of the musculo-spiral nerve also tells strongly against the theory of muscular antagonism. Unless the patient voluntarily uses the flexor muscles, the limb hangs in a state of rest, slight flexion and semi-pronation; and further, instead of the flexors contracting to their usual extent, the grasping power of the hand is greatly diminished from want of the sense of resistance by the extensors, so that at first one might think there was some actual loss of power in the flexors. The full effect of the extensor-paralysis is brought out by placing the upper arm in the horizontal position, and the fore-arm in that of flexion; then it is seen that extension of the elbow, wrist, and fingers is impossible.



Again, if the tendo-Achillis be divided, say, for overcoming the contraction of the calf muscles in oblique fracture of the tibia, the foot remains somewhat extended, whereas on the theory of antagonistic contraction it ought to become flexed (Volkmann).

Physiological contraction of the muscles alone, whether arising from central or peripheral irritation of the nerves, is rarely continued long enough to cause permanent deformity. It is seen in the "late rigidity" of the muscles following gross lesions of the brain; but here, too, the muscles undergo interstitial atrophic shortening as well.

(c) *Cicatricial and atrophic shortening* of the muscles and tendons.

The contraction of inflammatory lymph sometimes causes deformity of the joints—*e.g.* the thigh may be left permanently flexed as the result of psoitis. I have known talipes equinus follow deep-seated phlebitis in the calf of the leg.

If a joint—*e.g.* the knee or hip—be allowed to remain flexed for a long time, the muscles and ligaments will shorten up to the distance between their attachments by a process of interstitial absorption. In Pott's fracture one takes care to place the foot at a right angle with the leg before putting on a plaster casing. If this be neglected, some difficulty will be afterwards experienced in overcoming the resistance of the shortened muscles and tendo-Achillis and posterior ligament of the ankle-joint.

From what has been said it will be gathered that the chief causes of deformity lie outside the joints. In curvature of the spine a softened state of the bones and intervertebral discs greatly aids, and in fact may be the chief cause of, the permanent alteration in shape.

## CHAPTER LII.

## CURVATURE OF THE SPINE.

THE nature of the curvature depends (1) upon the disease which causes it ; (2) upon the distribution of the morbid process, whether it extends through the entire length of the column, or is limited to a certain region or part ; (3) upon the condition of the supporting structures, muscles, and ligaments ; (4) upon any extrinsic pressure or traction brought to bear upon the spine—*e.g.* in disease of the hip-joint the lumbar curve is exaggerated, together with a certain amount of rotation. Unilateral pleurisy may alter the natural curves, either during the effusion stage, or when this has passed away and given place to retraction of the same side of the chest wall.

Curvature in one region, such as that which follows Pott's disease, so alters the position of the centre of gravity that unless some means of *compensation* were provided the body would be in a state of unstable equilibrium. The effect of kyphosis in the dorsal spine is counteracted by secondary lordosis in the lumbar.

**Diseases causing curvature.**—1. Certain congenital defects. The lateral portions of the vertebræ may be imperfectly formed with or without coincidental defect in the ribs. The writer once saw a case in a girl aged about ten years, in which there was a firm nodule over the lower dorsal spine, apparently a cured spina bifida. Opposite this, two or three of the ribs on one side were fused into a solid plate. There was lateral curvature of the spine.

2. Caries (chap. xxxiv.).

3. Rickets (chap. xxii.).

## 4. Osteomalacia (chap. xl.).

5. In *aneurism* of the descending thoracic or abdominal aorta the bodies and contiguous parts of the neural arches of the vertebræ may be absorbed,

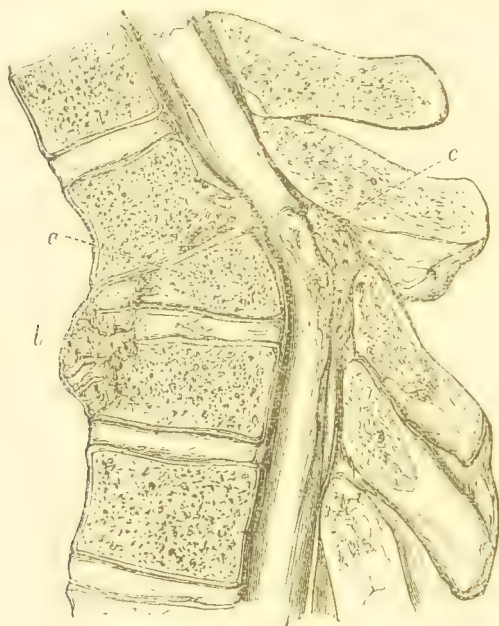


Fig. 50.—Fracture of the Spine.

*a*, Wedge-shaped mass, forming double-inclined plane, which has been driven backwards upon the cord, *c*; *b*, detritus of the anterior portion of the bodies of two vertebræ.

and this may lead to projection with lateral deviation of the spine.

6. *Chronic rheumatism (spondylitis deformans)* sometimes affects the vertebral column. In these cases the anterior common ligament is usually ossified, and the vertebræ are ankylosed. The spine is generally thrown into one uniform dorsal curve, and there are compensatory curves in the lumbar and cervical regions.

7. *Fracture* of the bodies of the vertebræ (Fig. 50). The history of an injury and the concomitant signs and symptoms suffice to make the diagnosis easy, although a long time may have elapsed since repair took place.

8. One of the most important forms is that known as scoliosis—"lateral curvature." It is doubtful where the fault lies in the first instance. Billroth believes that the primary cause is a weakness of the back muscles, and that the ligaments and discs are soft and yielding, and the bones affected with a "mild form of rachitis." It is most common in girls about the age of puberty, and in those compelled to sit or stand for a long time in a constrained position. This causes a sense of fatigue, and to gain relief the spine is supported unequally—*e.g.* by the hand against the object on which the patient is sitting. This gives the initial curve, which soon becomes exaggerated and permanent. If the dorsal curve is to the right, there is a compensatory lumbar curve to the left—compensatory, but not secondary, for in all probability both arise together. When the lateral curve is at all marked, a certain amount of rotation or twisting is also observed, the spinous processes being directed towards the concavity of the curve. After a time the bodies of the vertebræ become unequal in depth on the two sides, and sometimes they are more or less ankylosed by new bone. A single vertebra may be two or three times deeper on one side than the other.

#### SPONDYLOLISTHESIS.

Leishman, in his work on Midwifery, teaches that spondylolisthesis is a deformity in which the last lumbar vertebra slips downwards and forwards, and directly encroaches on the conjugate of the pelvic brim, and that it is due to disease of the spinal column. The researches of Neugebauer show that the above definition is too vague. According

to this observer, spondylolisthesis "is a deformity always produced during extra-uterine life, without the intervention of any original dyscrasia, or osseous affection of an inflammatory or specific nature (rickets, mollities ossium, caries, osteitis, etc.), by the physiological weight of the trunk, aided by certain

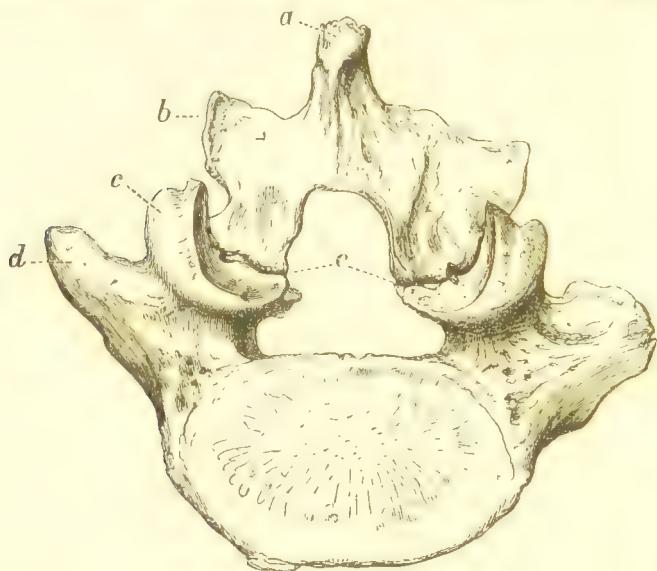


Fig. 51.—Fifth Lumbar Vertebra.

Showing complete cleft, *c*, through the intermediate portion, or that between the superior articular process, *a*, and the inferior articular process, *b*. *a*, spinous process; *d*, transverse process. The absence of signs of disease of the bone, and the want of symmetry between the two laminae of the neural arch, together with the bilateral disposition of the clefts, clearly show that we have to do with defective ossification, and not inflammatory absorption or fracture. In this case spondylolisthesis has not developed.

surgical predisposing causes, and that the deformity is not confined to the lumbo-sacral articulation of the spinal column, nor dependent on the age or sex of the patient." Prior to the enunciation of Neugebauer's hypothesis three theories had been advanced to explain the deformity: (1) that it was due to congenital hydrorachis; (2) that it was due to dislocation

of the lumbo-sacral joints ; (3) that it was the result of softening of the neural arch from caries. The first

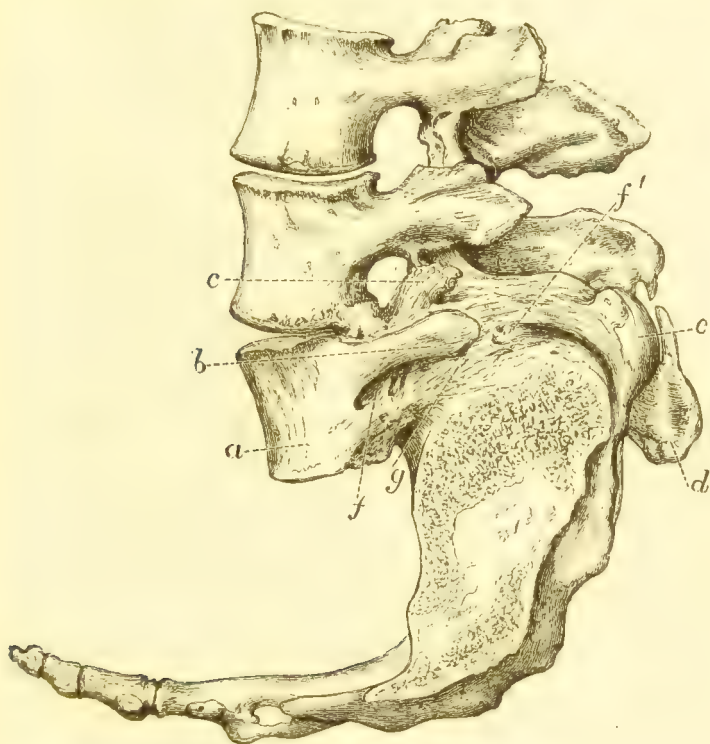


Fig. 52.—Spondylolisthesis.

*a*, Body of fifth lumbar vertebra, displaced downwards and forwards and ankylosed with the first sacral vertebra; it has carried with it the transverse process, *b*, and the superior articular process, *c*; *d*, spinous process of fifth lumbar; *c*, inferior articular process of same; *f*, *f'*, apertures of exit of anterior and posterior primary branches of fifth lumbar nerve. It will be seen that there is synostosis of the superior articular process of the fifth with the inferior articular process of the fourth lumbar; and of the inferior articular process of the fifth lumbar with the superior of the first sacral; *g*, "intermediate portion" of fifth lumbar vertebra, greatly elongated by the displacement. (University College Museum.)

two have little or no foundation in fact, and must be discarded; whilst the third involves a misinterpretation of facts.

In refutation of the last hypothesis it may be



remarked that caries of the spine very rarely destroys the neural arches to such an extent as to allow of displacement of the body of one vertebra upon another; it is more common for the neural arches to become ankylosed. Again in most of the known specimens of spondylolisthesis the vertebræ do not present the ordinary signs of antecedent caries, there is no caseation, and no abscess formation.

The two predisposing causes of the deformity, one of which is essential, are : (1) a congenital defect of ossification in the interarticular portion of the neural arch of the last lumbar vertebra; (2) fracture in the same situation, or it may be through the articular processes of the first sacral vertebra. The proximate cause is the weight of the trunk acting on the muscular and ligamentous supports, which it stretches.

It by no means follows that a solution of osseous continuity of the neural arches entails displacement of the superjacent column; this is an accident, as it were, and depends upon an inability of the soft structures to bear the strain thrown upon them; it is all the more likely to happen if there be enlargement of the abdomen (*e.g.* from pregnancy), for then the force of displacement is increased.

The deficiency or absence of ossification of the posterior segment of the fifth lumbar vertebra above referred to occupies the intermediate portion, or that between the superior and inferior articular processes. The result is that when the body of the vertebra is displaced forwards by the weight of the trunk, it carries with it the pedicle of the neural arch, the transverse process, and the upper articular process; whilst it leaves the posterior segment, consisting of the spinous process, the lamina of the neural arch, and the inferior articular process, fixed in its natural position.

It is obvious that a fracture through the interarticular portion, with defective formation of callus, would for all practical purposes be equivalent to a congenital arrest of development.

In extreme cases of the deformity (spondyloptosis) the body of the fifth lumbar vertebra overhangs the sacral promontory and encroaches on the brim of the pelvis. In the meantime, the interarticular portion of the neural arch necessarily becomes greatly elongated. Finally, the process is completed by synostosis of the displaced fifth lumbar with the first sacral vertebra (Figs. 51 and 52).

If displacement occurs after fracture through the articular processes of the first sacral vertebra, these processes, together with the entire fifth lumbar vertebra, are carried forwards until they are brought into contact with the anterior surface of the body of the first sacral vertebra. In support of the view that spondylolisthesis has sometimes a congenital basis, it may be stated that, in addition to the presence of fissures in the interarticular portion of the fifth lumbar vertebra, in certain cases the neural arches of the upper sacral are partly wanting.

Hergott considers that alteration of structure of the bone is the most important condition that gives rise to the deformity, and that traumatism is only an accessory cause. Neugebauer, on the other hand, maintains that the origin of the lesion, when not congenital, is always an injury behind the body of the vertebra, and that the osteitis and deformity are secondary.

## CHAPTER LIII.

## DEFORMITIES\* OF THE FOOT, KNEE, AND HIP.

**Talipes varus** is the most common variety of congenital deformity of the foot. The extension, if present, is due to a contracted state of the tendo-Achillis; the inversion, to a like defect in the tibiales, especially the tibialis posticus. The ball of the great toe is approximated to the heel, and the plantar fascia is shortened. The dorsum and outer border of the foot are more convex than natural. The tarsal bones are more or less wedge-shaped, the base of the wedge being outwards. In well-marked cases the astragalus appears as if partially dislocated outwards; but this appearance is really due to the inward obliquity of the os calcis, and to thickening of the head of the astragalus from friction. After the patient has learned to walk, the deformity increases, for the pressure exerted by the weight of the body is misdirected. Being greater on the inner portions of the tarsal bones than on the outer, growth is checked in the former direction; and as the movement of the bones one upon the other is greatly restricted, they may become ankylosed. The liability to this is increased by the irritation caused by walking and standing on parts ill-adapted to receive and transmit pressure. For the same reason callosities and subcutaneous bursæ are developed. The fore and middle parts of the foot are stunted in growth.

The foot is not really in a state of equinus, although the os calcis is displaced upwards and backwards, hence some pathologists prefer to designate the deformity simply talipes varus. The shortening of the

\* For an account of the causes of deformities see chap. li.

tendons and ligaments on the antero-lateral aspect of the foot prevents the fore-part of the latter from being directed downwards.

**Talipes equinus** is more often the result of injury or acquired disease than a congenital deformity. In the case from which Fig. 13 was taken, it followed cicatrization of a gunshot wound of the tendo-Achillis.

There is shortening of the calf muscles or the heel tendon. A dense callosity forms over the balls of the toes. The toes themselves are pressed and drawn upwards, so that they become partially dislocated on to the heads of the metatarsal bones.

**Pes planus** and **plano-valgus**. (Flatfoot.)—These deformities are usually met with in youth and early adult life. As regards their nature and cause, they are comparable to genu valgum and lateral curvature of the spine. As the consequence of long standing, the muscles and ligaments give way. Both arches of the foot, antero-posterior and intero-external, are lowered. “The head of the astragalus projects on the inner border of the foot, and the main deformity occurs at the astragalo-scaphoid joint. The instep falls in as well as down. In very severe cases the heel becomes raised, the tuber calcis being tilted up.” The calf-muscles, instead of simply elevating the heel, expend a good deal of force in causing movement at the astragalo-scaphoid articulation. The ligaments failing to support the strain upon them, the muscles are in a constant state of tension, and this causes *the symptom* complained of—viz. aching pain, referred to the instep or sole of the foot, or to the muscles of the leg.

**Talipes calcaneus** is one of the rarer forms of club-foot. It is usually congenital. Its occasional association with spina bifida has been referred to. The ankle-joint being in a position of acute flexion,

the tendons in front are shortened. If, at the same time, the foot is everted, the condition is known as *calcaneo-vagus*. Very rarely there is inversion—*calcaneo-varus*.

**Talipes valgus.**—In this affection the anterior part of the foot is everted, the displacement being effected at the mid-tarsal joints—astragalo-scaphoid and calcaneo-cuboid. The scaphoid is more or less carried outwards, and the prominence of the head of the astragalus thereby increased. There is shortening of the peroneal tendons. Commonly congenital, it is more often unilateral than pes varus. This deformity should not be confounded with flat-foot—spurious valgus.

**Pes cavus** is characterised by overarching of the foot, by extension of the first phalanges which are partially dislocated on to the dorsum of the head of the metatarsal bones, and by flexion of the second phalanges. The plantar fascia is shortened in proportion to the arching of the foot. If there is any paralysis or weakness of the leg muscles, pes cavus may be complicated by other forms of club-foot. The position assumed by the phalanges has been ascribed to paralysis of the interossei, and of the short flexor and adductor of the great toe, but no satisfactory proof has been given that this is the primary lesion. Pes cavus is an acquired affection.

**Hammer toe** usually affects the second digit. The first joint is extended, the second flexed, and the third commonly extended. A corn forms over the prominent first inter-phalangeal joint. The lateral ligaments are contracted and prevent extension of this articulation. Though said by some authors to be occasionally congenital, we believe that hammer toe is invariably acquired.

**Hallux valgus.**—As the name denotes, the great toe is displaced outwards. The head of the

metatarsal bone forms a marked prominence, and over it a bursa is formed, which becomes enlarged and thickened, constituting *bunion*. Suppuration in this bursa is of frequent occurrence, and by extension of the inflammation the contiguous joint is often involved.

**Congenital dislocation of the hip** may occur on one or both sides. The head of the femur is displaced on to the dorsum ilii, and consequently the trochanter is approximated to the anterior superior spine. There may be some adduction of the thighs, but there is generally very little rotatory displacement. The natural movements of abduction and external rotation are limited. The imperfectly developed head of the bone can be felt in its abnormal position. The acetabulum forms a mere depression or is absent. When the patient is standing, the pelvis is tilted forwards and there is marked compensatory lordosis of the lumbar and lower dorsal spine. If only one hip is affected, the spine will also be curved laterally. Locomotion is characterised by a peculiar rolling gait.

**Genu valgum** is the result of rickets in children, or continued strain upon the knees in youths and young adults, and more rarely in older people. The internal lateral ligament is stretched. The external and the biceps tendon are shortened. The inner condyle of the femur is said to be hypertrophied. What really happens is this: if the bending of the knee occurs after the bones are fully grown, the external condyle atrophies from pressure; if it comes on during childhood or youth, there is unequal growth of the two condyles: that of the outer is checked by the weight of the body transmitted through it, whilst that of the inner is less restrained. In rickets there is sometimes knock-knee of one limb and bandy-leg of the other. This is due to the way in which the child is carried by the nurse.



**Deformity from infantile paralysis.**—As before said, the normal foot, when in a state of rest, is partially extended and slightly inverted. The position is assumed and maintained by the weight of the foot and the *tone* of the muscles.

In infantile paralysis of marked degree the muscles are not only placed beyond the influence of the will, but there is a want of tone as well. The paralysis is rarely so extensive as to involve all the muscles of a limb; usually only a group of muscles or individual ones are affected. Now, although paralysis of one set does not at once cause displacement by the contraction of the antagonists, still, when the patient uses the foot, the want of power of support in the direction of the paralysed muscles must greatly influence the development of the deformity. This, however, is chiefly effected by other agencies. The foot being allowed to remain in the position assigned to it by its own weight, the soft structures (muscles, tendons, ligaments, and fasciæ) undergo an atrophic shortening, which serves to perpetuate the malposition. And, again, the weight of the body thrown upon the foot thus displaced aggravates the deformity or alters its direction. In extreme cases the affected foot is dragged after the other; and the inner border and fore part coming in contact with the ground, the extension is increased, whilst any original inversion is removed, or even replaced by eversion. If, on the other hand, the patient is able to use the limb fairly well, the equinus is still well marked, for the limb is always more or less stunted in growth, so that the sole of the foot cannot properly be brought to the ground in progression. The tendency is also to roll in or out according as the want of support is greater on one side or the other.

Thus the deformity from infantile paralysis is not a definite one. It depends upon several factors: the

weight of the foot, loss of tone in the paralysed muscles, secondary contractions and elongations of the muscles and ligaments, and the direction of pressure.

Infantile paralysis is due, in the majority, if not in all cases, to atrophy of the motor ganglion cells in the anterior cornua of the grey matter of the spinal cord, the result of anterior polio-myelitis. The sudden onset of the disease, accompanied as it not uncommonly is by convulsions, proves that the primary lesion is usually central; but it is possible that the nerve endings in the muscles are first affected in some instances. Unless the power of the paralysed muscles is restored within a short time (say a month or two) it will probably never be regained. Subsequent improvement in the contractility is then chiefly confined to fibres that have wasted from simple disuse.

When the paralysis is thoroughly established, the treatment consists of measures taken with a view of improving the nutrition of the limb in general, and the muscles in particular, such as electricity and massage; and in preventing and correcting deformities by the use of surgical apparatus.

**Other deformities** arise from congenital absence or contraction of muscles—*e.g.* the sterno-mastoid, arrested development of a bone or limb, persistence of a branchial cleft in the neck, etc.

There remains for description the pathology of spina bifida, cerebral meningocele and encephalocele, cleft palate, and extroversion of the bladder.

## CHAPTER LIV.

## SPINA BIFIDA.

SPINA BIFIDA is a congenital deformity that owes its origin to arrested development of the neural arches of the vertebræ.

The absence of development or of coalescence of the laminae allows the spinal membranes to protrude and carry with them the cord or nerve roots ; and in the case of the sacral spine, the beginnings of the nerve trunks. When no protrusion takes place the condition is termed *spina bifida occulta*.

The **tumour** is situated in the mid-line of the back. It is usually globular in shape, and about the size of an orange (Fig. 53). When oblong, the long axis is parallel to the spinal column. The extent in this direction depends upon the number of vertebræ involved.

The lumbo-sacral region (Fig. 53) is the most common seat of the affection—*i.e.* where the laminae of the neural arches are naturally late in completion ; but no part of the spine is exempt ; in fact, the entire neural canal may remain open.

It is not certain whether the arrest in development is always primary, or whether, in some instances at least, it may not be due to the pressure of fluid accumulated in the canal. The occasional coincidence of other congenital defects (*e.g.* talipes) seems to tell in favour of the former hypothesis.

The **wall of the sac** is composed of the integuments, the dura mater, and generally also the arachnoid, so that *the fluid* is contained in a cavity continuous with the subarachnoid, or “internal arachnoid” space. More rarely the sac is lined by the dura mater, and the fluid contained between

it and the arachnoid. These conditions are known as "hydro-meningocele."

In other cases the central canal of the cord is dilated, and the cord itself, or what remains of it,

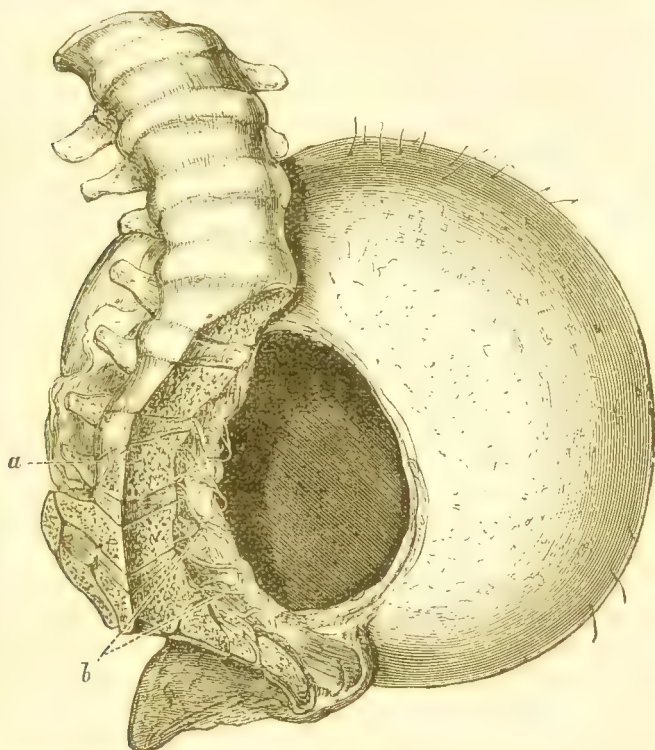


Fig. 53.—Lumbo-sacral Spina Bifida, from the Body of a Subject aged 29.

A portion of the sac has been removed to expose the nerves, *b*, which, after running for a short distance in the cavity, can be seen to enter its walls; *a*, bodies of vertebra in antero-posterior section. There is lateral curvature and rotation of the spine. (One-third natural size.)

spread out over the fluid (hydro-myelocoele, syringo-myelocoele), which in this instance, and also in internal hydro-meningocele, presents all the characters of cerebro-spinal fluid.

In many cases the tumour is a pure meningocele ; *i.e.* the sac contains neither spinal cord nor nerve roots.

The sac is unilocular, or divided into compartments by partial dissepiments. The skin over the tumour is sometimes very thin, and the sac of such tenuity as to rupture spontaneously.

Now and again a central dimple can be seen in the swelling marking the attachment of the spinal cord to the inner surface of the sac. It is clear that this, and the existence of hydro-myelocele, can only be met with in the dorsal, cervical, and lumbar regions, and rarely in the lower part of the last-mentioned situation ; for, before the tumour has reached any size, the cord has already receded in an upward direction, as the result of its growth being outstripped by that of the spine.

The **cerebro-spinal fluid** found in the sac is usually quite clear and colourless ; but it may be blood-stained from capillary rupture, or slightly turbid from inflammatory exudation.

Wilks and Moxon describe the process of secretion as an "irritative dropsy."

**Relation of the nerve-roots, nerves, and spinal cord to the sac.**—When the central canal is dilated, the cord may be spread out over the inner surface of the sac, or it may have entirely atrophied from the centrifugal pressure.

The cords of the cauda equina (nerve-roots) usually course through the centre of the sac, and "those which would naturally correspond to the vertebræ implicated in the tumour pass through the membranes to their distribution, while the lower ones return into the spinal canal" (Holmes). The nerve-roots may end in the wall of the sac, or they may be altogether absent. In the case before alluded to (page 329) there was no trace of the spinal cord, or of the roots of the nerves, in the neural canal.

**Associated deformities.**—The most common is chronic hydrocephalus ; next to this some variety of club-foot, usually talipes calcaneus.

Pressure applied to the hydrocephalic skull will sometimes increase the tension of the sac of the spina bifida ; but frequently the fluid collected in the ventricles is confined there by obliteration of the Sylvian aqueduct, or closure of the communication between the fourth ventricle and the subarachnoid space (Hilton). The latter condition I found in a case of lumbar spina bifida and chronic hydrocephalus.



## CHAPTER LV.

## CEREBRAL MENINGOCELE AND MENINGO-ENCEPHALOCELE.

*Cerebral meningocele and meningo-encephalocele* are the analogues of spinal hydro-meningocele and hydro-myelocele—*i.e.* the protrusion is either of the cerebral membranes alone, or of these together with the brain substance surrounding the ventricles. The opening in the skull is most commonly found in the occipital bone; it has been seen at the root of the nose, and the tumour mistaken for a nevus. More rarely the parietal and squamous bones are perforated, and, still more rarely, the base of the skull. In the latter case one would expect, on developmental grounds, to find the floor of the pituitary fossa absent.

It is very rare for the protrusion to take place at the cranial sutures. The tumour may or may not pulsate. This depends mainly upon the size of the aperture in the skull. It is more common in meningo encephalocele than in simple meningocele.

## CHAPTER LVI.

## CLEFT PALATE AND HARE-LIP.

IN the human being that portion of the upper jaw which carries the incisor teeth never remains separate from the rest, except as the result of arrested development and growth; but in the lower mammals—*e.g.* the dog—it exists throughout life as two distinct bones, called “intermaxillary” or “premaxillary.” And, even in congenital malformation of the palate and alveolar arch, the fissure never runs straight through in the middle line, but diverges near the anterior part of the jaw, and passes out between the portions of bone that carry respectively the incisor and canine teeth. That is to say, although the osseous continuity between the maxillæ and premaxillæ may be wanting, the premaxillæ themselves are always united into one piece of bone. In early fœtal life there is one large cavity, called the “nasobucco-pharyngeal”; this is subsequently partitioned off into four communicating compartments—the mouth, pharynx, and nasal fossæ—by the growth of two septa: the hard and soft palate and the septum nasi.

The roof of the mouth is completed by the union in the middle line of two laminae advancing from the sides of the upper jaw and palate bones, and the unossified structures behind. If this union fails to take place, cleft palate is the result.

It may consist merely of a bifid uvula, or extend through the soft palate, or through the whole or any portion of the hard palate; but, as before said, it never passes out in the middle line in front.

When the cleft reaches the posterior part of the

intermaxillary nodule it may bifurcate, and the two limbs of the fissure isolate that piece of the jaw. This is known as "double cleft palate." Single or double, when complete, it is almost invariably continuous with a split in the upper lip.

Cleft of the soft palate often exists alone ; so also hare-lip ; but cleft of the hard palate is rarely found without the velum being implicated.

As the roof of the mouth is wanting in the middle line, it is clear that the septum nasi, unless its lower border remains free, must deviate to one side or the other, in order to join the hard palate ; and this is what usually happens. •

When the cleft in the alveolar arch is double, the intermaxillary nodule can be swayed backwards and forwards, and the rudiments of the incisor teeth which it contains do not, as a rule, develop to the normal extent. This is one reason why, in operations for the cure of the deformity, the surgeon often elects to remove the piece of bone.

In *hare-lip* the fissure is almost always to the side of the middle line ; in the hare it is situated in the centre of the lip. It may be partial, or extend into the nostril. In the latter case the ala nasi is flattened, for the muscles draw it and the corresponding part of the lip outwards. Congenital median cleft of the lip is exceedingly rare.

## CHAPTER LVII.

EXTROVERSION OF THE BLADDER—ECTOPIA VESICÆ—  
HYPOSPADIAS.

IN ectopia vesicæ the anterior part of the abdominal wall and the anterior wall of the bladder are wanting. At birth they are usually present in the form of a thin membrane, which afterwards dries up and is cast off as a slough by the development of a ring of granulations at its periphery. When the defect reaches its highest grade, the proximal part of the umbilical cord, instead of being inserted as usual into the wall of the abdomen, is spread out as a membranous lamina continuous with the attenuated structures below, and, after ligature, separates with them.

The posterior wall of the bladder is thus exposed, and, having lost its support in front, is thrust forwards by the underlying viscera, so that, instead of a cavity, there is a bright-red elevation covered with mucous membrane.

The symphysis pubis, too, is often imperfectly developed, the pubic bones being united by a ligamentous band, and more or less everted by the resultant of two forces exerted by the adductors of the thigh and the oblique muscles of the abdomen.

The urethra is wanting. There is a groove on the dorsal surface of the penis, commencing at the lower part of the unnatural opening in the bladder. When this opening is confined to a small aperture above the pubes, the condition is known as *epispadias*.

In ectopia vesicæ the urine constantly escapes from the exposed orifices of the ureters, and, coming

in contact with the skin of the abdomen, causes considerable irritation.

The transition from skin to mucous membrane is imperceptible.

**Hypospadias** is due to imperfect development and union of the lateral lappets that go to form the normal urethra. It may extend the whole length of the penis, but it is generally confined within a short distance from the end of the glans. In the former case the scrotum may be cleft. Instead of the urethra being wanting in its anterior part, as described above, there may be a small opening at the peno-scrotal angle or in the perinæum.

## CHAPTER LVIII.

## FATTY DEGENERATION OF ARTERIES, AND ARTERITIS.

MANY terms more or less misleading have been used in the description of arterial pathology; *e.g.* *atheroma*, which is really the consequence of precedent inflammation and degeneration, is often used to indicate the substantive disease. In this work the morbid processes and their results will be described as they occur separately or in combination.

**Fatty degeneration** is met with under two conditions: (1) As a primary and solitary change; (2) as the sequel of inflammation.

(1) *Primary fatty degeneration* is best seen as it affects the endothelial and subendothelial layers of the internal coat. It is most common in the first part of the aorta, and although it increases in frequency as age advances, it is by no means rare in early life. It causes small, slightly elevated yellowish patches. The fat granules are for the most part arranged in groups, still retaining the outline of the stellate endothelial cells (Fig. 9, A). The tissue beneath may be quite healthy; so that these cases have no clinical significance.

The fat granules are absorbed or swept off by the blood stream, and new cells replace the old ones.

But the degeneration may be more widespread in the coats of the vessel. This is very constant in senile decay. Rounded and bead-like collections are seen in the middle coat, following the course of the elastic laminæ and muscular fibre-cells.

(2) *As the sequel of inflammation*, fat molecules are never absent. The greater part of the contents of "atheromatous abscesses" is composed of them.

**Acute endarteritis.**—Primary diffuse suppurative or phlegmonous arteritis is unknown. The disease



in question affects chiefly the large arteries, especially the aorta. In some cases there is a history of syphilis, but in most the cause is obscure.

The inner surface of the vessel has an irregular greyish, semi-translucent appearance, as if melted gelatine had been sprinkled upon it, and had then set. Some patches are yellowish from fatty change. Calcification is rare. When cut into, the inflamed spots are found to be firm and gelatinous-looking. The swollen intima may be many times thicker than

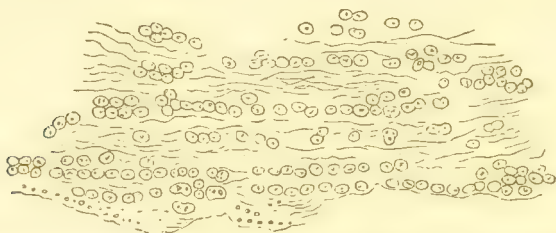


Fig. 54.—Section of the Tunica Interna of the Aorta, in a Case of Acute Endarteritis. (Cornil and Ranvier.)

natural. The middle coat is more or less involved, but never to the extent as in chronic arteritis (atheroma). Under the microscope the diseased structure is found to be infiltrated with small cells, which lie parallel to the laminae (Fig. 54). The cells are derived from the vasa vasorum. In old cases the cells may almost entirely have disappeared, leaving a dense cartilaginous homogeneous substance, and a few fat molecules.

The most pronounced case I have seen was in a subject with aneurism of the aorta and both popliteal and the right femoral arteries. The entire aorta was studded with hard, smooth, grey elevations, from the size of a pin's head to that of a pea.

**Chronic endarteritis.**—Atheroma. This disease begins in the deeper portion of the internal

coat. Small cells accumulate in the midst of a homogeneous or granular matrix of exudation matter and softened intima. This goes on until there is bulging into the vessel, which here appears of a dull, yellowish-white colour. Should the inflammation stop at this stage, its products will be partially absorbed subsequent to fatty degeneration, and the remaining portion of the intima will come in contact with the middle coat; or, if the latter has been destroyed, the internal and external coats will blend, forming together a slightly depressed fibrous cicatrix. But, meanwhile, lime-salts are frequently deposited, and then a brittle calcareous plate serves as a mark of the previous arteritis. During the slow progress of the inflammation the cells are transformed into fat, and this again into its chemical derivatives; so that a so-called *atheromatous abscess* is filled with degenerating cells, granular débris, carbonate of lime, and crystals of cholesterine and the fatty acids, and, possibly, hæmatoidin crystals. If the pellicle, which bounds the atheromatous focus next the lumen of the vessel, gives way, the contents of the cavity are washed out by the blood stream, and the resulting excavation is termed an *atheromatous ulcer*. The base of the "ulcer" usually calcifies, or becomes fibrous. The calcareous plates above mentioned sometimes split away at the margin, and pigment is deposited beneath them from the blood which coagulated as it trickled through the fissures. They also act as foreign bodies, and cause extensive thrombosis. They may be detached, and give rise to embolism.

Dr. Handfield-Jones has drawn attention to occlusion of the small arteries of the brain by the fatty débris set free from atheromatous patches. Such occlusion must evidently lead to degenerative changes in the nerve elements. It is strongly suggestive of Bright's disease.

**Arteritis deformans.**—In *old people*, chronic inflammatory and degenerative changes are widely spread. Homogeneous thickening of the internal coat, atheromatous patches, calcified plates in the large arteries, and calcified rings in the small and medium-sized ones, may all be found in the same subject. On the whole, these senile arteries are dilated, but the inner coat may be so thickened as to cause serious obstruction to the blood current, and cause thrombosis and gangrene. Like varicose veins, the arteries are elongated, so that they are thrown into permanent curves.

**Syphilitic disease of the arteries.**

(1) Syphilis, by impairing the nutrition of the tissues generally, may lead to early senile changes, fatty degeneration and atheroma.

(2) But there is a specific syphilitic inflammation of the arteries more or less acute in its progress. It begins in the internal coat (Fig. 56), but the entire thickness of the walls may be converted into a glassy or slightly fibrillated substance containing small cells. In the arteries of the brain there is a good deal of exudation into the perivascular sheaths as well, so that the lumen is narrowed from swelling within and compression without.

It is one of the chief causes of cerebral thrombosis. The softened state of the vessels renders them very liable to aneurismal dilatation. In the case of the aorta and other large arteries the inner coat is enormously thickened and uneven.

Atheroma and calcification are less common than in senile disease.

**Arterio-capillary fibrosis** enters largely into the pathology of granular gouty kidney. It is not limited to that organ, but is widely distributed throughout the various tissues (skin, nerve-centres, etc.). The walls of the vessels are thickened by a hyalin-fibroid material (Gull and Sutton) and hypertrophy of the

middle coat (Johnson). The lumen of the vessels is diminished. The obstruction it causes induces hypertrophy of the left ventricle of the heart.

Other causes of arteritis need only be mentioned. They are chronic alcoholism and rheumatism.

**Inflammation of the middle and external coats.**—It is doubtful if this occurs as a primary disease. The middle coat may be destroyed by inflammation reaching it from either side. The external is involved in like manner, but it especially suffers in periarteritis.

**Periarteritis** consists of an inflammation of the external coat and its areolar sheath. They rarely escape altogether in acute and chronic endarteritis. It has been shown that in syphilitic disease of the cerebral arteries the perivascular sheaths are extensively infiltrated; in fact, several vessels are occasionally embedded in a continuous mass of exudation.

In thrombosis of the deep veins the periphlebitis extends to the companion arteries. Not only may the areolar sheath and external coat of an artery be softened by inflammation until bulging takes place, as in aneurisms of branches of the pulmonary arteries skirting phthisical cavities, but the tension upon the vasa vasorum may be so great that the entire thickness of the wall of a large artery may slough away—*e.g.* the femoral in spreading perilymphatic abscesses of the groin.

Periarteritis, by causing canalisation, serves as a dangerous check upon hæmostasis in wounded vessels; it prevents contraction and retraction of the artery within its sheath.

**Progressive obliterative arteritis.**—Cases are met with now and again in which an artery (*e.g.* the brachial) and its branches gradually become rigid and eventually pulseless, and this, too, without any assignable cause such as injury, syphilis, embolism, or senility. The obliteration of the lumen usually

commences in the distal vessels and creeps slowly upwards. The symptoms are severe pain, blueness alternating with pallor of the skin, wasting of the structures supplied by the artery, and sometimes peripheral gangrene. Opinions are divided as to the share thrombosis takes in the morbid process. Some observers—*e.g.* Bryant—believe that the arteritis is consequent on coagulation of the blood; whilst others, including Friedlander, hold that the disease is a primary inflammation of the arterial coats. In Gould's case,\* the patient was only nineteen years of age when he noticed the fingers of the right hand getting dark in colour; in the end, the brachial artery up to within an inch of its origin became pulseless and much diminished in size. Gould says, "the slow progress and the marked induration of the vessels before pulsation was lost were sufficient to exclude simple thrombosis, especially in the arteries of the upper limb of a young man of this age."

It is not a question whether pressure, embolic plugging, syphilitic disease, or atheroma, may cause obliteration of the lumen of an artery, as that point is well established; but whether there is such a condition as "primary progressive obliterative arteritis." The balance of evidence certainly supports this theory.

The absence of widespread gangrene is remarkable, considering the extent of occlusion of the vessels.

**Raynaud's disease: local asphyxia; symmetrical gangrene.**—In 1862 M. Raynaud published an account of certain phenomena pertaining to the peripheral circulation, which he designated "*local asphyxia*" and "*symmetrical gangrene*." These were shown to be dependent on disturbance of the vasomotor apparatus.

The variation of the external signs is explained by the degree and duration of the vascular spasm, which is the essential factor in the morbid process. In

\* *Lancet*, Feb. 16, 1884, p. 296.



addition to the instability of the cutaneous nerves there is probably, in some cases at least, an exaggerated excitability of the vaso-motor grey matter of the spinal cord. In the simplest case the arterioles, capillaries, and veinlets are contracted, so that the skin is pale, cold, and numbed. To this condition, commonly known as *dead finger*, Raynaud applies the name "*local syncope*," in contradistinction to "*local asphyxia*," in which the parts are of a blue, purple, or even blackish hue. In the latter state the spasm is to a great extent confined to the arterioles, through which little blood passes, the current in the capillaries at the same time being sluggish, since the back pressure from the veins is incompletely overcome by the diminished force of the arterial stream. In extreme cases the contraction is so great and sustained that the tissues become gangrenous. The fingers and toes are the parts most affected. There is a great tendency to bilateral symmetry. The patients are often quite young (*gangrène juvénile*). The attack usually occurs in cold weather. "Paroxysmal attacks of hæmoglobinuria have been observed in some of the recorded cases, and appear to be distinctly associated with the affection."

**Gangræna cutis hysterica.**—Riehl,\* F. Ehrl,† and Féré‡ have described cases of spontaneous gangrene of the skin in hysterical patients. The gangrene presents itself in single or multiple patches. In most of the recorded cases severe pain was experienced in the part which afterwards became necrotic. Various nervous symptoms have been found associated with the local disease. In Riehl's patient, a young girl, there was anæsthesia over certain areas of the skin, anæsthesia of the cornea, contraction of the field of vision, and hyperæsthesia of the ovarian region.

\* *Wien. klin. Wochenschrift*, 1893, p. 826.

† *Wien. klin. Wochenschrift*, 1894, p. 330.

‡ *Comp. Rendus des Séances de la Société de Biologie*, 1894.



## CHAPTER LIX.

## ANEURISM.

**Varieties of aneurism.**—Aneurisms are either traumatic or pathological (spontaneous). The term traumatic is limited to those cases that form directly as the result of injury. Many pathological aneurisms can be traced to some local strain, but of such a degree that had the vessel been healthy it would not have given way.

**Diffuse traumatic aneurism.**—Blood escapes from the wounded vessel into the interstices of the surrounding structures, and in the direction of least resistance—*e.g.* in the intermuscular planes and beneath fasciæ. The distending force is too great to allow of an adventitious sac of inflammatory tissue being formed. Thus, a traumatic aneurism of the popliteal artery may spread up the thigh, or the blood from a ruptured axillary fill the arm-pit. The only treatment likely to avail is ligature of the artery on each side of the wound or amputation. The cause is usually a stab or rupture during the attempt to reduce an old dislocation.

**Sacculated or circumscribed traumatic aneurism.**—These are either simple or arterio-venous. In the former case the sac is variously constituted. (1) It may be formed by a yielding cicatrix, after the wound in the artery has closed; this can only happen when the artery is small, or the opening in it very minute. (2) It may consist of a protrusion of the internal, or internal and middle, coats through a wound in the external (hernial aneurism). (3) It may be formed of the external coat, the internal and middle having been lacerated by

ruising against a bone (Fig. 55). (4) The sac may be purely adventitious, all the coats having been divided; in such cases the blood escapes very slowly, the inflammatory induration around sufficing to prevent a rapid extension of the aneurism.

**Arterio-venous aneurism** results from the simultaneous wounding of an artery and neighbouring vein—*e.g.* the brachial and median basilic in blood-letting at the head of the elbow. If the inflammatory exudation caused by the injury fixes the vessels in contact, the blood will pass directly from the artery to the vein, and the latter will become dilated and tortuous, and will have its walls thickened from the irritation of the strain (*aneurismal varix*).

But the cementing lymph may yield and form a sac between the two vessels (*varicose aneurism*). This is the more dangerous variety, since the sac is usually thin, and the tension upon it is more concentrated than on the resilient walls of the vein. The pulsation, bruit, and thrill are also stronger than in aneurismal varix.

**Pathological or spontaneous aneurisms** are divided into true and false. *The sac* of a true aneurism is said to be formed of all the coats of the vessel, but on pathological grounds the distinction is not well founded; for the disease of the vessel ere it allows of

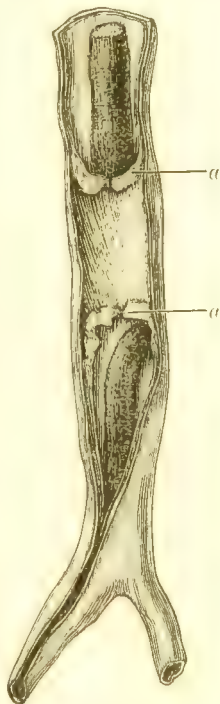


Fig. 55.—Occlusion of the Popliteal Artery following Rupture of the Internal and Middle Coats, *aa*, which are curled up within the vessel.

The space between the pointers is formed by the external coat only, and is free from coagulum. Clots are seen attached to the curled-up coats.

dilatation has caused at least the middle coat to disappear. Syphilitic and idiopathic arteritis (atheroma) are the causes of the weakness of the vessel. In chronic arteritis the deeper layers of the internal coat and the middle coat are destroyed, so that what remains of the internal coat is blended with the external, and both are so diseased that their natural structure cannot be made out. Frequently, also, the

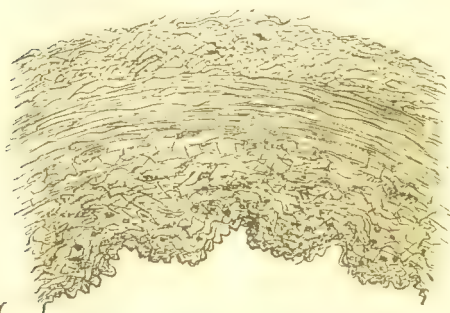


Fig. 56.—Section through Femoral Artery, just below an Aneurism.

a, Tunica intima, ruged and greatly thickened.

aneurism develops at the seat of an atheromatous ulcer, when nothing but the external coat remains. In most cases the irritation kept up causes the sac to be thickened by the interstitial deposit of lymph and by inflamma-

tory condensation of the areolar tissue outside. From want of elasticity the sac continues to dilate in spite of its thickness. But usually there comes a time when the dilatation is so rapid that the sac is greatly thinned, and finally it gives way (*ruptured aneurism*).

The **clot within the aneurism** is more or less laminated. The first-formed laminae lie at the bottom of the sac, but do not reach its neck, whereas the more recent ones do. This is explained by the increase in size of the aneurism. The lamination is due to variations in the rate of coagulation, or to a succession of coagulations, so that the superficial layer of the last deposit has undergone change by the time coagulation sets in again. Moreover, there is

sometimes an alternation of pale and coloured clots (Fig. 57), the former consisting chiefly of fibrin, the latter of coagulated blood. The darker strata become paler as time goes on, from disintegration of the red corpuscles and diffusion and absorption of the hæmoglobin.

The laminae can be easily separated from one another, and the outermost from the sac of the

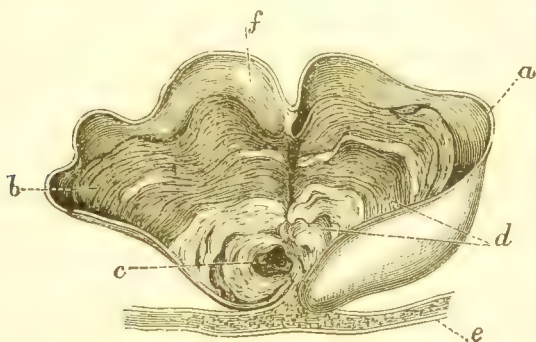


Fig. 57.—Aneurism of Middle Cerebral Artery. (Natural size.)

*a*, Sac of aneurism laid open, and showing the laminated clots, *b* and *d*, within; *c*, black coagulum; *e*, artery; *f*, space left by clot shrinking from hardening in alcohol. The older peripheral part of the clot was of a dark reddish-brown colour, interspersed with two pale streaks. The more recent part of the clot was of a pale fawn colour, with the exception of that last formed, which was black.

aneurism. Irregular softening cavities occasionally form in the clots of large aneurisms, and in this way fragments may be detached and plug the vessel beyond, and possibly cause gangrene. If the laminae are loosened at their free borders blood passes into the clefts and, meeting with little resistance between the layers, separates them more or less. Then the interlaminar spaces fill with dark coagula. This is another explanation of the alternation of pale and coloured clots; but in such cases the disposition is very irregular.

From natural causes, or as the result of treatment,

the blood in the sac may coagulate *en masse*. However extensive the lamination, the final obliteration is always effected by simple coagulation, which extends into the artery on each side of the aneurism.

The greater part of the clot becomes absorbed, the aneurism shrinks, and is ultimately converted into a nodule of connective tissue. The danger from softening of the coagulum in the aneurismal sac is not great, for by the time it occurs the adjacent part of the artery is pretty firmly plugged.

Under the microscope, scrapings from the laminated clot show fibrin filaments, fatty débris, granular and crystalline hæmatoidin, and plates of cholesterine.

**Changes outside the aneurism.**—The irritation from expansion of the sac sets up inflammation around it. This causes a thickening of the sac at the expense of the tissues, which undergo gradual absorption, partly from inflammatory softening, partly from diminution in the blood supply, through continuous pressure. Where the irritation is slight, the exudation organises, so that in the same specimen new tissue formation and atrophy may be seen side by side (Fig. 8). No tissue is able to withstand the expanding force of an aneurism; thus the sternum, ribs, and vertebræ become excavated in aneurism of the aorta. Cornil and Ranvier assert that rarefying osteitis caused by the pressure of the sac is the sole cause of the absorption of bone. No doubt it is an important factor, but simple atrophy from sustained pressure must not be forgotten. In some cases of rapid enlargement of the aneurism the inflammation in and around the sac goes on to suppuration. This inevitably causes rupture, unless in the meantime coagulation has taken place in the aneurism and artery beyond; but even then the clot will probably disintegrate, and the final result be the same.

The superficial veins of the part are generally



dilated, from obstruction to the current through the main trunk. There may also be œdema.

The resistance offered to the blood stream as it passes through the aneurism increases the tension in the side branches of the artery, and so opens up the collateral circulation. For this reason gangrene of the leg is less likely to follow ligature of the femoral for a large or medium-sized than for a small aneurism. Hence also the value of compression preliminary to ligation.

The existence of a spontaneous aneurism is evidence of arterial disease, which renders the vessels rigid and slow to expand.

The pulsation of the artery on the distal side of the aneurism is usually weak ; at the same time, the artery may be fuller than normal, from blood entering it in many collateral streams.

The bruit produced in the sac may be conducted for a considerable distance along the artery ; *e.g.* in the case of a femoral aneurism it may sometimes be heard in the tibials. This is an important diagnostic sign between aneurism and a pulsating malignant tumour, in which the bruit arises in many vessels and away from the main trunk.

**Spontaneous cure** may arise from (1) gradual coagulation within the aneurism ; (2) rapid coagulation due to (*a*) some local condition—*e.g.* acute inflammation of the sac—or (*b*) a general disease in which the arterial pressure is diminished, and possibly the composition of the blood altered ; (3) detachment of clot from the aneurism and consequent plugging of its orifice, or the artery beyond.

**Varieties of pathological aneurism.**—These are named : (1) From the composition of the sac—true and false ; (2) from their shape—sacculated, fusiform, cylindrical, and racemose or cirroid ; (3) from their primary origin—syphilitic, embolic ; (4)



from some peculiarity in size, course, or distribution—miliary, dissecting, etc.

**Fusiform aneurisms** only attain a large size in the case of the aorta. They are not uncommon in the popliteal arteries. There is uniform dilatation of the entire circumference of the vessel. They generally yield at one part, and become sacculated.

**Cirsoid aneurism.**—Racemose aneurism.—Aneurism by anastomosis consists of a dilatation of a connected series of arteries, or of a single artery and its branches. It is most common in the scalp and orbit, but it is met with in other parts—*e.g.* the buttock. The vessels are enlarged in every way, they are lengthened, tortuous, and varicosed; their walls are thickened, and their lumen increased. Billroth believes these aneurisms to be of inflammatory origin, since they sometimes follow injury, and their walls contain many new-formed cellular elements. In one case I found two circumscribed aneurisms of the internal carotid, one just outside the skull, and the other in the cavernous sinus; and also uniform dilatation of the ophthalmic artery and its branches. There was protrusion of the eye-ball. The patient had shortly before been kicked by a horse on the side of the head. Ligature of the common carotid failed to cure or arrest the disease, and death ensued from cerebral congestion. In another patient all the arteries of the scalp were dilated, and the occipital was as large as the little finger. By their pressure they had hollowed out the cranial bones into furrows.

**Embolic aneurisms** are mostly intracranial. Vegetation from the cardiac valves arrested in the cerebral vessels set up a localised arteritis. Dilatation takes place at these spots. Such aneurisms are relatively common in children.

**Miliary aneurisms** are found in the cerebral vessels. They are small, often no larger than a

pin's head. Some are microscopical. They are usually multiple, and are met with in the pia mater and in the substance of the brain. Atheroma or syphilis is the cause. By their rupture they cause apoplexy, but previous hæmorrhage or thrombosis may in some cases be the exciting cause of the dilatation, the obstruction in front throwing strain on the diseased arteries.

**Dissecting aneurism.** — An "atheromatous abscess" bursts; blood is forced between the middle and external coats, or more probably between the layers of the middle coat (Peacock). The channel thus formed opens again into the lumen of the artery, when it meets with a softening focus or another ulcer; or it ends in a sacculated aneurism of the internal or external coat, and then it obstructs the artery, or ruptures externally. Dissecting aneurism is not met with in the small arteries. It is most common in the aorta.

## CHAPTER LX.

## LIGATURE AND TORSION OF ARTERIES.

WHEN an artery is tied the internal and middle coats are cleanly divided, but they do not curl up as in torsion (Figs. 58, 59). The



Fig. 58. — Common Carotid Artery of a Child, on which a Catgut Ligature was applied three days before death. The clot has been removed.

"a" points to the furrow made by the ligature, which completely severed the internal and middle coats of the vessel.

external coat is thrown into folds, and the convolutions are welded together by the ligature. The injury inflicted on the vessel induces coagulation, which extends on each side to the next collateral branch. The clot is pyramidal, with its base at the seat of ligature. At first it fills the artery for some little distance, but it shrinks as the contracting fibrin expresses

the serum. This allows the blood to pass between it and the vessel wall, and further deposit takes place, until the plot is more or less spirally laminated. When fully formed it is firmly adherent to the wall of the

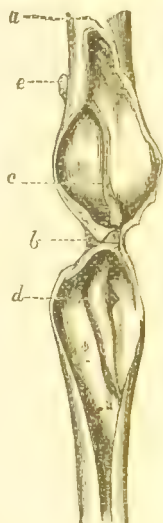


Fig. 59. — Portion of Femoral Artery examined on the fourth day after the Application of a Ligature.

"a," Cardiac extremity; "b," division of the internal and middle coats by the ligature; "c," proximal thrombus; "d," distal thrombus; "e," origin of profunda femoris. (After Maneg.)

vessel. Within a fortnight it is quite pale from solution and diffusion of the colouring matter. The proximal is said usually to be larger than the distal clot.

The so-called *secondary clot* consists of inflammatory lymph and cells derived from the vasa vasorum under the irritation of the ligature. The exudation joins the primary clot about its base and sides, and is, in fact, incorporated with it. Organisation ensues, and the lumen of the vessel is obliterated by dense cicatricial tissue. Most observers agree that the clot is partly absorbed and partly organised. Cornil and Ranvier say that it is wholly absorbed, and that the young vascular connective tissue that fills the vessel is entirely derived from an ingrowth of granulations from the inflamed walls. The process is exactly the same as in occlusion by spontaneous thrombi (Fig. 62).

As the vasa vasorum run for a short distance within the sheath before penetrating the coats of the vessel, it is clear that the vascular supply to the arterial walls will be somewhat defective immediately on the distal side of the ligature. Moreover, as the collateral circulation is being established the tension is constantly being raised on the distal side and lowered on the proximal. The better nutritive supply and the greater quiescence explain why the artery on the proximal side of the ligature is more firmly occluded than on the distal; and why in the case of secondary hæmorrhage the blood more often issues from the distal portion.

**Cutting through of the ligature.**—What happens is as follows: The portion of external coat gripped by the ligature is killed outright, and is cast off like any other slough when the wound is septic. This is effected by a true process of ulceration. A layer of granulation tissue forms on either side of the ligature holding the necrosed portion of the artery. The latter is not seen on examining the loop of the

ligature, for it disintegrates whilst being set free. But if the ligature remain aseptic, it will become absorbed or embedded. When an artery is ligatured at some distance from an aneurism on the proximal side, the artery is obliterated at two points: (*a*) at seat of ligation, (*b*) just above the aneurism. The intermediate portion of the artery is not obliterated. It may shrink somewhat, or it may retain its normal calibre.

**Torsion.**—When torsion is applied to the cut end of a large artery the internal and middle coats curl up and form a valve-like septum which, in a healthy vessel, as large even as the femoral, is of itself sufficient to arrest bleeding. The outer coat is twisted over the others, and thus gives additional security against hæmorrhage. When the arteries are much diseased, the coats are more or less blended and rigid, and the elastic tissue to a great extent destroyed. Such a condition renders torsion inapplicable. In rare cases the inflammatory and degenerative changes are so pronounced that a ligature will sever all the coats of the vessel. Acupressure should then be resorted to. When a large artery is torn across by accident the result is practically the same as if it had been twisted. Short of complete rupture of an artery, the inner and middle coats may be lacerated and partially detached whilst the continuity of the outer coat is not interrupted (Fig. 55).

## CHAPTER LXI.

## VARIX.

**Definition.**—Varix is a dilatation of the veins the coats of which are thickened, and the lumen is widened and lengthened. There is usually considerable pouching opposite the valves, and here the attenuated walls are liable to rupture.

**Causes.**—The exciting and sufficient cause is the strain from obstruction to venous circulation; but besides this there is often a predisposition to the disease from inherent weakness of the coats of the veins. The obstruction may be temporary—*e.g.* the pressure of a tumour; recurrent, as in repeated pregnancies; or permanent, as in aneurismal varix.

**Consequences.**—(1) Thrombosis and phlebitis, as in inflamed piles; (2) ulceration (in the leg); (3) atrophy of structures—*e.g.* the testicle, from varicocele, this arising partly from pressure and partly from deprivation of properly oxygenated blood.

**Usual seats of varix.**—The lower extremity; the prostatic, spermatic, and hæmorrhoidal plexuses; and the neighbourhood of arteries (aneurismal varix and varicose aneurism).

The veins of the lower extremity suffer more than those of the upper: (1) Because the force of gravity acts more continuously against the direction of the venous blood-stream; (2) because contraction of the muscles of the lower limb is greater and more continuous, and the blood from the deep intermuscular veins is forced into the superficial ones to a corresponding extent (advantage is taken of this compensatory dilatation in the operation of venesection); (3) accidental pressure upon the inferior vena cava



and iliac veins is more common than upon the superior vena cava and innominates.

**General pathology.**—By some the initial change is described as inflammatory, whereas others look upon it as a mere *fibroid substitution*—that is to say, the normal elements of the coats are to a great extent replaced by a low form of connective tissue, the same as is seen in nutmeg cirrhosis of the liver. In both cases the increase of fibrous tissue follows chronic obstruction to the flow of blood through the veins. Formerly these changes were considered as inflammatory; now it is the fashion to speak of them as “cirrhoses,” “scleroses,” or “fibroses.” It is difficult to differentiate the two, for whilst the usual signs of inflammation are not marked, because they are very slight and spread over a long period, the final result is the same as one of the typical terminations of undoubted chronic inflammations, that is, the production of cicatricial tissue.

Those who reject the inflammatory theory assert that the stagnation in the veins prevents their coats from being properly nourished (for if the current through the veins is obstructed, it is clear that the tributary vasa vasorum cannot discharge their contents with natural freedom). They say that the deoxydised blood suffices for the nourishment of connective tissue, but it is not equal to the support of more highly organised structures such as muscular fibre.

We refuse to accept this as the entire explanation; (1) because the effect of strain, which is traumatism, long continued, perhaps, but none the less traumatism, is ignored; (2) because the areolar tissue around the veins is much condensed and thickened by material that is more vascular and corpuscular than normal.

There is degeneration from chronic starvation, and inflammatory thickening from strain.

As before said, there is general increase in the

width and length of the veins. They dilate in spite of the thickness of the walls, for the intravascular pressure is raised *ab initio*, and the altered structure is less able to bear the strain thrown upon it. On account of the great elongation the veins become curved in a remarkable manner. Then follows varicosity: (1) because the obstruction tells most at the seat of the valves; (2) because the blood stream is diverted from its rectilinear course, and its impact is greater on the convex side of each curve as seen from without, like as a river hollows its banks here and there in its winding channel.

**Histology.**—On opening a varicosed vein the inner surface looks rough from a longitudinal striation. Under the microscope this is seen to be due to coarse bundles of connective tissue interlacing with the elastic fibres of the inner portion of the middle coat and greatly obscuring them. The muscular fibres at first undergo a compensatory hypertrophy, and on section lengthwise of the vein appear, if stained with logwood, as pale circles with dark central spots—muscular fibre-cells and their nuclei respectively. Eventually, the muscular and elastic elements are so overrun by fibroid overgrowth that nothing can be seen but a partly fibrillated, partly homogeneous, scar-like tissue, in which are embedded granules of *pigment*, scattered, or in groups. The vasa vasorum are dilated and their coats thickened.

**Calcification** may take place (1) in the form of plates in the walls of the veins; (2) as *phleboliths* in the interior, and probably in the remains of blood-clots that have formed about the valves. They are most common in the prostatic veins.

The **valves**, which are merely reduplications of the intima, suffer in the same way as the walls of the veins; that is, they become thicker and less pliant.

Their function is soon lost, for they undergo cicatricial contraction, whilst the lumen of the vein is increased. In the end they shrink to mere caruncles, or even disappear. Should one of the ampullary dilatations of a greatly distended superficial vein rupture, blood will issue from the proximal and distal sides of the opening. The more dangerous bleeding often comes from the proximal side, for the valves no longer suffice to support the column of blood above them (it may be from the vena cava downwards); and the weight of this column causes a continuous downpour, which may end fatally in a few minutes, unless checked by pressure or by elevation of the limb.

**Changes around the veins.**—The areolar tissue in which the veins lie is congested and infiltrated with inflammatory exudation. The exudation becomes indurated and firmly united to the veins, so that the latter are more or less fixed. This accounts for the feeling of a shallow trench in the skin and subcutaneous tissue when the finger is pressed firmly along the course of a dilated vein. The fixation of the veins, together with the thickening and rigidity of their walls, prevents them from collapsing when wounded, and so favours hæmorrhage.

*Pigmentation* of the skin and cellular tissue results from capillary extravasation and transudation of red corpuscles. The discoloration around varicose veins of the leg is often very marked.

The localised thinnings that lead to rupture are due to extreme distension of varicosities behind the valves, and to want of support from without. Thus, we often find that the superficial part of the vein, together with the atrophied skin over it, is not so thick as the deeper portion of the vein alone.

## CHAPTER LXII.

## EMBOLISM.

EMBOLISM, or the conveyance and subsequent arrest of foreign bodies in the blood-vessels, begins either on the arterial or venous side of the circulation.

The **sources of arterial embolism** are, vegetations from the cardiac valves, clots dislodged from the sac of an aneurism, the contents of an "atheromatous abscess," and calcified plates and thrombi from the walls of the large arteries.

**Venous embolism** usually results from the detachment of clots. The entrance of air in the case of wounds of veins within the "dangerous region" is well known, and the symptoms of these cases are probably the consequence of plugging of the pulmonary vessels by aërial emboli.

Less common causes of embolism are, aggregations of pigment granules, portions of new growths swept off from the interior of vessels which they have invaded, pus from the bursting of an abscess, and parasites.

**Fat embolism.**—Liquid fat may find its way into the circulation in the following circumstances:— (1) In cases of fracture of bone, especially when compound, and the central medulla broken up; (2) in severe contusions or lacerations of adipose tissue; (3) in rupture of a fatty liver; (4) in septic inflammations. The liquid fat set free enters into the circulation through the open vessels, chiefly the veins, possibly the lymphatics. Its entrance is aided by increase of pressure whether due to blood extravasation, inflammatory exudation, or the force of an injury. In pyæmia the fat emboli being contaminated with

the poison of the disease, are likely, in addition to their mere mechanical effects, to set up fresh centres of inflammation. The emboli are arrested in the capillaries and perhaps small arterioles. The symptoms produced are referable chiefly to the lungs, where they may give rise to hæmoptysis, and other consequences of obstruction to the pulmonary circulation. Fat embolism is stated to be sometimes fatal, death being due either to plugging of the cerebral or pulmonary vessels. The fat can be best demonstrated in the capillaries by osmic acid staining. The fat which has entered the circulation has been shown to be largely eliminated by the kidneys.

**The effects of embolism.**—The immediate effect of embolism is anæmia of the part supplied by the obstructed vessel; but inasmuch as there is in most cases a free collateral circulation, blood makes its way in by the side channels, and if the embolus be rigid, only partial occlusion may occur; this also happens when it is arrested at the point of bifurcation of an artery, for then it may not be large enough to completely close either branch. Where the collateral circulation is but little developed, as in the intermediate-sized vessels of the brain, a venous reflux ensues, which soon leads to stasis, since the pressure in front is no longer overcome by the force of the current in the artery.

The embolus, acting as a foreign body, causes coagulation of the blood upon it. This reaches to the first collateral branch, where its progress is arrested, provided the anastomosis is sufficiently free. When it is defective, and especially if a number of contiguous branches of the same vessel be occluded, the blood stagnates, and the whole area becomes the seat of secondary thrombosis. This, however, takes some time to become complete; and in the meanwhile, the walls of the capillaries, suffering from impaired



nutrition, readily allow the passage of blood-corpuscles, and probably many capillaries actually rupture, the more so as the strain upon them is increased by the distension of the congested vessels around. This is what is termed *hemorrhagic infarction*.

The size of the embolus will depend upon its source and its consistence. If it be derived from a calcified cardiac vegetation, or an atheromatous plate from an artery, it will withstand the impact against the walls of the vessels. Should it consist of a softened venous thrombus it will very likely be broken up in its onward course, the several portions being scattered in an embolic shower. A vessel may not be completely blocked by an embolus, but, later, occlusion may ensue from coagulation of the blood upon the embolus.

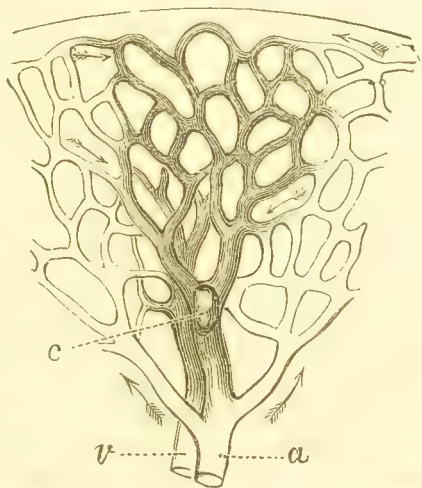


Fig. 60.—Diagram of Embolic Congestion of the Lung.

*a*, Small artery plugged at *c* by an embolus; *v*, small vein filled with a clot, which extends as far as its trunk; the shaded portion of the capillary network is the area of distribution of the artery, in a state of passive congestion, and about to be the seat of a hemorrhagic exudation. The arrows show the collateral channels through which the abnormal turgescence is effected. (Kindeisich.)

The **effect upon the wall of the vessel** will vary as to the nature and duration of the plugging. If the embolus consist of a healthy thrombus it may be quickly absorbed, leaving the structure of the vessel intact; or it may become adherent and organise,



the lumen in this way being permanently obliterated. A rough body, such as a calcified vegetation, will irritate mechanically, and cause thickening of the coats of the vessel. A portion of a tumour may infect the part at which it is arrested, and form the centre of a fresh growth. Lastly, from whatever source, a septic embolus invariably leads to acute inflammation and necrosis.

**Paths of transit.**—Since as a rule the emboli are comparatively small, and tend to take a direct course, it is clear that the periphery of an organ will be more often the seat of the lesion than the centre; hence in the lungs the posterior bases suffer most. Inasmuch as they are carried along the main course of the blood stream, they will pass into the thoracic aorta rather than travel through the innominate, carotid, and subclavian orifices. So, again, they will be found in the left carotid, renal, and iliac arteries more often than in the right. In a few instances—*e.g.* in the middle cerebral, coronary of the heart, and branches of the celiac axis—their course is almost at a right angle to that of the parent vessel.

When the embolus starts on the venous side of the circulation (which it usually does, and in the form of detached clots) its arrest commonly takes place in the lungs, and the pulmonary capillaries filtering the blood prevent the distribution of the solid particles in other organs or tissues. But these capillaries are comparatively large, and are still further dilated in any disease that raises the pulmonary pressure; hence it is not very uncommon to find that some of the smallest emboli make their way through the lungs and left side of the heart into the aortic system. Still there is no doubt that many of the metastatic abscesses found in other organs than the lungs in pyæmia are the sequel of primary thrombosis, and not of embolic infarction.

In cases where the abscesses are found only in the liver (excluding those of portal origin), the same explanation seems more probable than that which supposes the emboli to have passed from one or other cava into the hepatic veins, or, in other words, against the blood current; although in severe obstructive disease of the chest the systemic venous pressure may be so increased as to become positive.

Thrombi, or portions of new growth set free from branches of the portal vein, will be conveyed to the liver, and there probably entirely arrested.

**Hæmorrhagic infarction** has been already adverted to. It consists of secondary thrombosis, starting from an embolus and extending through a part or the whole of the area supplied by the obstructed artery, and, as a result, exudation from the vessels, extravasation of blood from rupture of capillaries, and the formation of an intense hyperæmic, inflammatory zone around (Fig. 60). These infarctions are mostly wedge-shaped, for the vessels divide again and again, the area of distribution gradually widening (Fig. 61).

The pathological changes that ensue depend: (1) Upon the size of the infarction; (2) upon the freedom of anastomosis; (3) upon the nature of the plug, whether it be simple or septic. If the embolus be small and the collateral circulation free, a quick absorption of the clot in the thrombosed vessels is the usual result, and the part is thus restored to its healthy condition. If the embolus be large, but not septic, the area of infarction is too wide for the thrombosed vessels to be cleared before fatty degeneration takes place in the tissues (including the walls of the vessels themselves) cut off from their blood supply. In this case the colouring matter of the coagulated and extravasated blood is discharged from the corpuscles, and, with the more fluid portion, is absorbed by the surrounding vessels, leaving a firm

yellowish-white putty-like mass, composed of degenerated tissue and blood-clot, closely resembling a syphilitic gumma, for which, indeed, it is sometimes mistaken; but a gumma is harder, and is not conical in shape, nor is it surrounded by such an intensely congested zone. This mass, in its turn, is gradually reduced in size by absorption of the debris resulting from the disintegration, until at last a contracted,

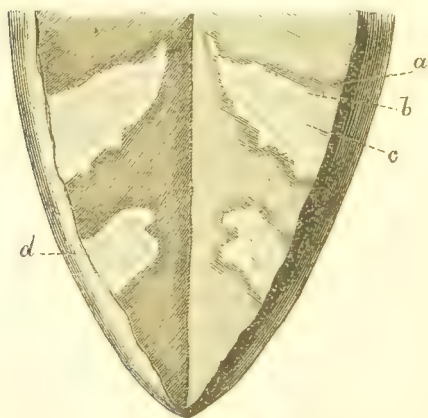


Fig. 61.—Embolic Infarction of Spleen.

The wedge-shaped masses are partially decolourised. This is farther advanced at the periphery, *b*, than in the central portion, *c*. Each block is surrounded by a zone of hyperemia, *a*; *d*, outer surface of the organ.

puckered cicatrix, infiltrated, perhaps, with lime-salts, and containing some pigment granules, and, in the case of the brain, hæmatoidin crystals, is all that remains.

If the infarction start from a septic embolus, whether it be large or small, the intense irritation will invariably lead to the formation of a circumscribed metastatic abscess,

the contents of such abscess consisting of inflammatory exudation, disintegrated clot, and broken down tissue.

**General effects of embolism.**—The original embolus may be so small, or it may crumble into such minute fragments, that only the capillaries are blocked, and unless in this case it be septic, or some vital organ, such as the brain or heart, be involved, it will probably lead to no further trouble. On the other hand, it may be of such a size as to cause almost instant death, as when large vegetations from a cardiac valve find their way into the cerebral vessels, or a

thrombus loosened from an iliac vein plugs the trunk or main branches of the pulmonary artery.

Besides the immediate danger in such cases, and the almost certainty of death in septic embolism of the internal organs, there are others of great gravity.

Gangrene of the leg may follow the accidental plugging of the popliteal artery by a clot from an aneurism of the same vessel, the opening up of the collateral circulation being too long impeded by the rigidity of the diseased vessels. (*Vide* page 367.)

*Aneurism of the cerebral arteries* often owes its origin to the irritation of an inflammatory embolus; the walls softening from inflammation yield at first on the proximal side of the obstruction. The source of the embolism is frequently detachment of vegetations from the cardiac valves after rheumatic fever, especially if the endocarditis be "infective or ulcerative." This explains the apparent anomaly of pathological aneurisms occurring in children.

## CHAPTER LXIII.

## THROMBOSIS AND PHLEBITIS.

THROMBOSIS is a clotting of the blood in the vessels. Concerning the coagulation of blood drawn from the vessels, it can be shown that it is due to the interaction of three bodies, fibrinoplastin, fibrinogen, and a ferment resulting in a combination of the two former ; that is to say, fibrin, which does not exist in the blood, comes to be formed under certain ascertainable conditions as the latter becomes solid. There is, so to speak, a constant antagonism between two sets of influences—between those that favour and those that retard or prevent coagulation. Amongst the former are : (1) Rapid motion, as in whipping, or, in other words, multiplication of points of contact. (2) Exposure to not living matter. Amongst the latter we have : (1) The addition of a third of the bulk of some neutral salt ; (2) a temperature of about 32° Fahr. ; (3) moderate heat ; (4) the abstraction of hæmoglobin ; (5) the absence of a neutral salt, whose presence, according to Schmidt, is essential to coagulation.

The proximate cause of coagulation is believed to be a ferment set free by the death and disintegration of the white blood-corpuscles.

Now blood, whilst in the vessels, is known to clot more easily when the current is slowed, just the reverse of what is found in shed blood when whipped ; but this is only an apparent anomaly, for slowing of the current means a diminution of the nutrition of the coats of the vessel, and this brings it to a state of lower vitality, and so the question of slowness or rapidity of motion is resolved into that of contact with foreign matter. As to the so-called ferment, the

conditions under which the consequences of its action are brought about in the living body cannot be doubted; and it is here that the antiseptic treatment of wounds shows itself to advantage, for when decomposition of the discharges is allowed to take place unchecked, how much more likely is thrombosis to extend beyond the immediate seat of injury, how much more likely a rapid softening of the clot, and, as a natural consequence, detachment of emboli rife with mischief, by reason of their mechanical action, and still more from their being saturated with septic matter.

**Causes of thrombosis.**—To gain some clue to an answer to the question, Why does the blood coagulate in the vessels? the diseased states not seldom complicated with this result must be considered. Amongst others, the following cases have occurred in the experience of the author:—thrombosis of the cerebral veins in phthisis and marasmus; of the veins of the upper limb in septicæmia from compound fracture of the jaw; of the pulmonary artery, fatal when the aseptic wound from amputation of the breast was all but healed; of the varicose veins of the lower extremity; of the axillary and femoral veins in post-parturient women; of the axillary vein in rheumatic fever; of the brachial artery from bruising. To these may be added plugging of the cerebral arteries in syphilis, capillary thrombosis in cancerum oris, and as the explanation of the multiple openings in carbuncle (Billroth); not forgetting those cases where coagulation is intentionally induced, as in the ligation of arteries and the operations for the occlusion of varicosed veins.

There are two diseased conditions, one of the vessels, the other of the blood, that act as the proximate causes of thrombosis.

Slowing of the blood current from weakness of the heart is also an important factor.



**Causes found in the blood.**—The blood may be so vitiated that it can no longer maintain its fluidity, from (*a*) increase in the fibrin factors, (*b*) excess of excrementitious matter, from defective elimination, as in gout, or the introduction of infective material, as in septicæmia.

**Causes in the vessels.**—Alteration in the structure of the vessels from defective nutrition can be explained in three ways: 1. Those conditions that tend to slow the blood current; such as (*a*) varicose veins; (*b*) atheromatous and calcareous arteries, and aneurismal sacs; (*c*) obstruction from without, as by ligature, or the pressure of a tumour, or inflammatory exudation, causing great capillary tension; (*d*) venous reflux into capillary areas, from embolism of the arteries feeding them; (*e*) feeble cardiac action, either from fatty degeneration, or acute myocarditis, or a deficient output of energy from want of the proper amount of stimulus, as when a person of active habits is suddenly confined to bed by an accident. 2. The influence of inflammatory states, such as occur in subacute and chronic arteritis; and simple degeneration of the vessel walls. 3. The immediate effect of injury—that is, before there is time for inflammatory action to supervene.

The state of nutrition of the tissues to be nourished must necessarily affect the quality of the blood, and so indirectly lead to clotting.

**Relation of thrombosis to phlebitis.**—The microscopical changes found in thrombosis have been studied chiefly in the veins, where coagulation is much more common than in the arteries. For a long time phlebitis was considered the cause of thrombosis; at a later period there was a strong tendency to reverse the sequence, and with it the cause. Some pathologists even went so far as to deny the existence of an endo-phlebitis, contending that the so called

inflammation of the vein was in reality a peri-phlebitis affecting the adventitia and the surrounding tissue.

The argument that (1) the signs of inflammation along the course of a vein during life, (2) the redness of the intima, and (3) the ofttime puriform contents of a vein found on dissection, justified the conclusion that the disease was primarily and essentially phlebitis, was traversed on the following grounds:—(1) That a periphlebitis would explain the symptoms; (2) that the discoloration of the intima was due not to hyperæmic injection, but to simple staining by hæmoglobin set free from the corpuscles of the thrombus; and (3) that the material regarded as a mixture of pus and blood was partially decoloured, softened, disintegrated clot. But even allowing, as one must, that the physical signs above referred to were often misinterpreted, it does not follow that under no conditions does phlebitis precede thrombosis; nay more, there is ample evidence that such is frequently the case.

Primary thrombosis results (1) from septic emboli detached from disintegrating clots steeped in the products of bacterial putrefaction; (2) from emboli derived from the site of an infective inflammation, and therefore containing micrococci and alkaloidal bodies developed by their fermentative action; (3) free infective organisms circulating in the blood—*e.g.* in splenic fever.

The initial lesion may be considered to commence in the walls of the vein (1) when the latter is implicated by extension of inflammation from the adjacent structures—*e.g.* in cellulitis; (2) when it is injured mechanically with or without the presence of an open wound.

In a septic wound we have the conditions that underlie alike primary thrombosis and primary phlebitis; the former arising from contamination of

the blood by septic fluids soaked through the walls of the veins, the latter by the irritative action of these fluids on the veins themselves.

In so-called gouty phlebitis it is a disputed point whether thrombosis is the precursor of phlebitis, or *vice versâ*.

**Signs of phlebitis.**—When a subcutaneous vein is inflamed the signs are very manifest: redness of the skin over it, a hard cord with nodular swellings—*i.e.* the occluded vein still further thickened by the exudation of lymph around, the nodules marking the position of varicosities and the valves. It may be noted that as the clot shrinks or is absorbed more quickly from the internodal portions, small, hard lumps for a time remain, which in certain situations (*e.g.* Scarpa's triangle) may be mistaken for indurated glands. Then there will be œdema from obstruction of the current, and as the clotting and inflammation spread, the point of greatest intensity of pain will shift along the course of the vessel from day to day. It is by no means rare in patients with shattered health, for abscesses to form here and there around the veins that have become plugged. When the thrombus is septic, suppurative periphlebitis is the rule rather than the exception. It is a matter of some difficulty to dissect out these veins from their bed of lymph, which, later, is either organised, leaving the vessels blocked, or is absorbed, the normal condition being restored. When the deep veins are thrombosed there is often no redness of the skin, but the pain, œdema, and distension of the superficial veins point to the pathological changes going on (*phlegmasia alba dolens*).

When diffuse coagulation occurs in a main artery and its companion vein it usually begins in the artery: and, again, diffuse thrombosis of an artery is more frequently attended by that of the vein than *vice versâ*,

for when a vein is plugged, the nutrition of the walls of the contiguous artery is not necessarily much interfered with, but when the circulation in the artery is stopped, the vasa vasorum that supply the walls of the vein are also obstructed, and so a starvation of that part of the vein ensues, and this causes a diminution of vitality and consequent coagulation. At the same time it is likely that the inflammatory state outside the artery thrombosed may have some share in effecting the clotting in the neighbouring vein; besides, it is certain that something more than a simple failure of nutrition is at work, else how comes it that coagulation occurs in only a small proportion of the cases of degenerated vessels?

**The fate of the clot.** — Absorption, organisation, disintegration, and suppuration are the secondary changes occurring in the thrombus. Except in the case of septic thrombosis, where, if life be sufficiently prolonged, softening always ensues, *absorption* is perhaps the most common event. This is often seen in varicose veins that have been treated by simple compression, where after temporary occlusion the channel is re-established. Absorption may proceed by a gradual disintegration of the central part of the clot, the blood current passing through the latter, which in the end is entirely removed; or wasting may slowly progress from the periphery until only a fine filamentous cord of fibrin entangling white corpuscles (for the red ones break up early) is all that can be seen; and this finally disappears, leaving the lumen of the vessel completely free. Embolism in these cases is prevented by the clot in the main vessel being kept in position by offshoots into the tributary branches. After absorption is complete, staining of the intima persists for some time. *Disintegration* occurs in all septic thrombi, and occasionally in large simple clots where the blood supply is defective; thus,

in aneurisms that have been consolidated by rapid coagulation of the blood *en masse*, as in those of the popliteal artery treated by Esmarch's rubber and cord, it is by no means rare to find fluctuation after some days. It would be a grave error in practice to mistake this liquefaction in the centre of the clot for suppuration. Such cases require great care in the after-treatment, for it is difficult to tell the exact extent of the softening, and injudicious manipulation might dislodge a portion of the contents and force it into the trunk and branches beyond. Although a healthy clot projecting into a vein may be broken off by the impact of the blood stream, by careless handling, or by movement on the part of the patient (particularly flexion of the hip when the ilio-femoral vein is thrombosed), still such an event rarely happens, unless there be loss of consistence and tenacity from disintegration. It is the existence of this softening in pyæmia that explains the very frequent occurrence of embolic infarctions and abscesses.

**Organisation of the clot.**—In some text-books it is stated that after phlebitis the vein sometimes shrinks to a "fibro-cellular cord." This is misleading, for it is doubtful if direct adhesion of the opposite sides of the intima ever takes place. The correct explanation seems to be that changes occur in the clot that end in its complete organisation, and the connective tissue thus formed undergoing cicatricial contraction and blending with the intima of the vessel already thickened and vascularised, an impervious cord is the result. When a clot is about to organise, the red corpuscles disintegrate, and the colouring matter set free is in great part absorbed. Granules of pigment, however, persist for a long time. Leucocytes find their way into the interior, elongate, ramify, and anastomose. The fibrin, according to some, degenerates and is removed, whilst others state that

it fibrillates and helps to form connective tissue. Capillary blood-vessels develop (*vide* Organisation of inflammatory deposits, page 16) and join the main channel, and also the vasa vasorum (Otto Weber). Fig. 62 represents a section of a carotid artery occupied by young vascular connective tissue. The case

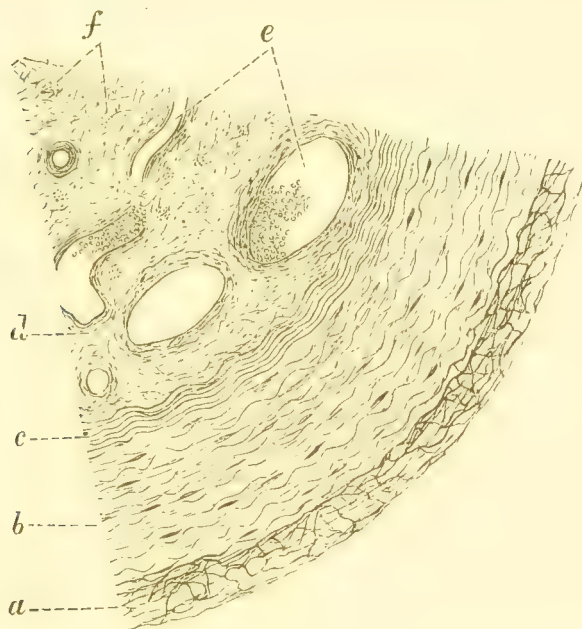


Fig. 62.—Organised Thrombus in Common Carotid Artery, from a Case of Aneurism of the Aorta.

*a*, Tunica adventitia; *b*, tunica media; *c*, tunica intima; *d*, vascularised thrombus united with inner coat of artery; *e*, vessels of thrombus; *f*, groups of pigment granules.

was one of aortic aneurism, in which the orifice of the left carotid was closed. Thrombosis gradually spread from below until the whole vessel was occluded. Sections at different levels demonstrated the successive changes from recent coagulation to complete development of fibrous tissue. According to Cornil



and Ranvier, it is the irritation by the clot that sets up the endophlebitis, which consists of a proliferation of the endothelium, exudation of lymph cells, and the formation of vessels which penetrate the thrombus; they say that as the latter is absorbed the inflamed intima fills up the lumen of the vein, and if its blood-vessels dilate at the same time, a cavernous tissue results, but as a rule the channel is permanently obliterated.

**Suppuration** may occur in septic thrombi. The leucocytes are derived from the vasa vasorum of the walls of the vessel softened by inflammation, and possibly also from the blood that bathes the thrombus, whilst many, no doubt, collected in the vessel before coagulation. The migratory corpuscles, whatever their source, mingle with the liquefied débris of the clot. The pigment of the disintegrated corpuscles gives to the whole a buff, reddish-yellow, or chocolate colour.

**Coagulation before and after death.**—Clots formed during the last few hours of life in the right side of the heart and pulmonary artery, and in the systemic veins, are often as much the result of dying as the cause of death. They cannot for certain be told from post-mortem coagula, since sufficient time has not elapsed for secondary changes to set in. In both cases (unless post-mortem coagulation is very rapid) the lower portion of the clot is deep violet or quite black, and the upper of a pale buff or yellow colour, from subsidence of the red corpuscles. The whole is tough and elastic, and is smooth on the surface and not firmly adherent to the vessel which contains it; in fact, it never completely fills the vessel. Undoubted ante-mortem clots, on the other hand, are more brittle, often breaking with a granular fracture, are more uniformly pale from decolorisation, and are for the most part adherent.

The difference is well marked where an old softened venous thrombus has been detached and swept into the right side of the heart, and there formed the centre of fresh coagulation.

Ante-mortem thrombi are composed of concentric laminae, for the coagulum first filling the vessel shrinks like any other blood-clot, and thus allows a lazy current to trickle between it and the vessel wall. A succession of alternate clottings and contractions goes on until the vein is finally blocked.

The clots always contain a large number of leucocytes, which accumulate in the vessel during the preliminary stages of slowing and stasis.

Dr. Bastian has described a capillary obstruction by an aggregation of white corpuscles.

## CHAPTER LXIV.

## ACUTE ORCHITIS AND EPIDIDYMITIS.

ACUTE inflammation of the testis may be excited in one of three ways: (1) By direct injury; (2) by metastasis; (3) by extension of the process from the urethra and spermatic cord.

In the two former cases the body of the testis is usually involved before the epididymis. With regard to metastasis in the specific disease, "mumps," it is open to question whether the orchitis is not the direct result of the poison of the fever rather than a transference of inflammation from one gland to another, between which there is no developmental, anatomical, nor physiological relationship. The fact that orchitis follows in the wake of parotitis is no proof that it is the consequence of it. The correct pathology is probably explained by saying that the poison of mumps has a much less affinity for the testis than the parotid, and thus affects it more rarely; and when it does, at a later period.

Gonorrhœal *epididymitis* is in most instances the result of spreading of the inflammation from the urethra along the vas deferens and lymphatics to the epididymis, for the cord is often enlarged, indurated, and tender, and the vas deferens still more frequently. Its late occurrence can be accounted for by the time taken by the inflammatory process to extend through such a lengthy tract, and the simultaneous subsidence of the urethritis may be explained by supposing that the severe affection of the epididymis acts as a derivative through the functionally associated nerves.

As regards the theory of metastatic origin I know of no facts sufficient to warrant its acceptance, and

those facts consistent with it—subsidence of the urethritis with the onset of epididymitis, and the apparent immunity from attack of the vas deferens and cord in certain instances—are capable of a more satisfactory explanation. In uncomplicated gonorrhœal epididymitis I believe the vas deferens never escapes, and although it may be but slightly enlarged, it is always more or less tender. Of course the testis or epididymis, or both, may become inflamed through a blow or squeeze of the organ in a patient suffering from gonorrhœa, and then one would not expect to find the vas and cord affected, except at a later period, by the inflammation spreading upwards. It is then primarily a case of traumatic and not gonorrhœal epididymitis.

*Blood poisoning* rarely leads to orchitis, either by way of embolism or by the direct action of invisible infective matter circulating in the blood. I have once seen pyæmic suppuration in the sac of an old hydrocele of the tunica vaginalis.

*Gout* is another disease that occasionally affects the testis, one, too, in which there is liability to recurrence of the inflammation.

Orchitis is a good instance of exudation beneath a resisting membrane. This, taken with the natural sensibility of the organ, explains the severe aching pain. The hyper-sensitive nerves are compressed by the effusion on the one side and by the inelastic fibrous tunic on the other.

The lymph is effused first between and then into the seminiferous tubes. The epithelium becomes swollen and granular, and its cells proliferate. As the inflammation subsides healthy cells replace those cast off, the same as in desquamative nephritis. Whilst the formative activity of the epithelium is increased, the functional power is diminished.

In some cases, especially in orchitis, the sequel of

mumps, the functional power is permanently lost, and as the old degenerated cells are removed by absorption, the whole organ atrophies. This must be partly attributed to the specific effects of the poison upon the gland epithelium, in the same way that the hair follicles and papillæ sometimes waste after the exanthematous fevers, causing baldness. This explanation is based on the fact that the intensity of the inflammation is no certain guide to the final result, repair or decay, although probably cicatricial contraction of the inflammatory exudation constricts and obliterates some of the vessels.

As the inflammation spreads from the seminal tracts to the surrounding tissues, it causes thickening of the cord, œdema of the scrotum, and *acute hydrocele*. The tunica vaginalis rarely contains more than two ounces of fluid, usually less. The fluid is highly fibrinous, and readily coagulates when withdrawn, differing in this way from that of simple chronic hydrocele, which, though rich in fibrinogen, does not often gelatinise spontaneously. It is generally absorbed as the orchitis subsides. By far the most frequent termination of orchitis is in resolution. Sometimes the inflammation becomes chronic; supuration is the exception.

## CHAPTER LXV.

## CHRONIC ENLARGEMENTS OF THE TESTICLE.

THESE are designated by the common term *sarcocoele*, which simply means a fleshy swelling. Several distinct affections are embraced by the definition, which can only be justified by long usage.

The varieties of sarcocoele usually enumerated are : (1) Simple ; (2) syphilitic ; (3) scrofulous or tubercular ; (4) malignant ; (5) cystic. The first three are essentially inflammatory ; the fourth, with some exceptions, includes the fifth (page 410).

**Simple sarcocoele** may be mild from the first, or the sequel of one acute or several subacute attacks.

The body of the organ is uniformly enlarged, hard, and not very tender or painful. When the epididymis is affected, which it almost invariably is, and firmly adherent to the testis proper, the swelling of the globus major and minor may give a nodulated outline to the mass.

There may be some *fluid* in the tunica vaginalis, but frequently the cavity of the latter is obliterated. The *cord* is somewhat enlarged from dilatation of the vessels. The existence and amount of induration depend to a great extent upon whether it was implicated or not by the original inflammation.

As a rule only one testicle is affected, but when the inflammation is set up and perpetuated by the continued irritation from a stricture of the urethra, both may be involved.

The lymph, instead of breaking down to form an abscess or softening cavity, or being absorbed, organises to dense fibrous tissue. The cut surface presents a greyish semi-translucent aspect, with perhaps a dash



of yellow, from fatty degeneration of the epithelium and the wandering cells. It is homogeneous, or streaked with bands of connective tissue. The microscopical appearances vary with the extent and duration of the inflammation.

When the disease is of long standing nothing may be seen but an imperfectly fibrillated structure, with a few indifferent cells and fat granules scattered over the field. Most of the blood-vessels having been obliterated, it appears almost extravascular, unless the remaining ones be shown up by artificial injection.

In more recent cases one recognises in some parts minute clefts or alveoli filled with granular debris and degenerated cells; but there is no regularity of disposition, for whilst in one place the exudation is homogeneous or faintly striated, and practically devoid of vessels and corpuscles, in another it is more vascular and cellular.

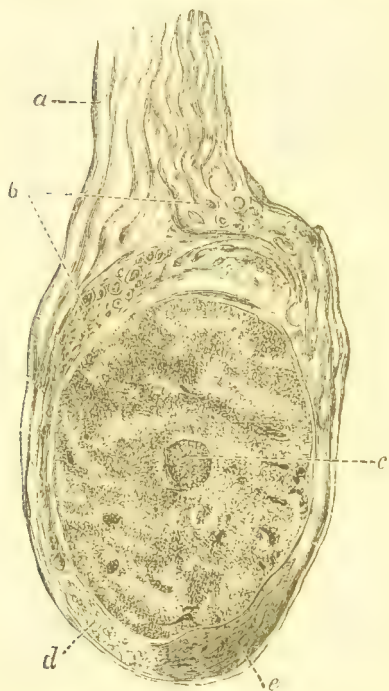


Fig. 63.—Syphilitic Orchitis.

*a*, Vas deferens; *b*, blood-vessels distended with gelatin; *c*, small gummatous deposit; *d*, portion of epididymis; *e*, inflammatory thickening of tunics. The cord is enlarged, but not indurated. The cavity of the tunica vaginalis is obliterated, except at the upper part of the testicle. The entire organ is infiltrated with inflammatory exudation, but the blood-vessels are still everywhere pervious. (Reduced one quarter.)

**Syphilitic sarcocoele.**—Syphilitic disease of the testis presents itself under two forms: diffuse orchitis and localised gummy tumour. In the former case

the enlargement of the organ is uniform, and the surface consequently remains even. In the latter, whilst one part of the testis retains more or less its natural anatomical features, the other is occupied by a hard swelling, which projects as a rounded nodule or craggy mass, giving an irregular outline to the swelling.

Again, the exudation may in the first instance be confined to one region, and afterwards become diffused, showing that the minute pathological changes are essentially the same in both cases. In the circumscribed gummatous mass the inflammation may be said to be concentrated. The vessels, too, are extensively obliterated, by pressure of the effusion without and by thrombosis within. The latter process is enhanced by the diseased condition of the vascular walls, which, by causing thickening and irregularity of the intima, diminishes the lumen and obstructs the flow of blood. The obliteration of the vessels, coupled with the inherent low vitality of the exudation, explains the subsequent degeneration.

Fig. 63 represents a testicle removed post mortem from the body of a man who, at the time of his death by accident, was the subject of well-marked tertiary syphilis. The vessels have been injected with carmine and gelatin. In the centre of the organ is a rounded nodule of very firm consistence, which is clearly a gumma in process of development. It is quite vascular, but, from its induration being greater than the surrounding tissue, it has shrunk less by the action of the spirit in which the specimen is preserved, and is consequently raised above the general section surface. The entire body of the testis is enlarged, but the epididymis is not appreciably affected. On microscopical examination of the nodule, the tubules were found to have been destroyed and replaced by inflammatory cells. At some distance

beyond, the tubules could still be recognised, embedded as they were in richly corpuscular exudation, but the epithelium had lost its natural characters. The vessels of the cord are enlarged, but they are quite distinct, being lightly held together by flocculent areolar tissue; hence there is no induration. There is no hydrocele. The other testicle was quite healthy.

This preparation may be taken as being fairly representative of syphilitic orchitis, diffuse and gummatous. It is interesting as showing the vascularity of a gumma in the early stage of formation.

**Modes of termination of syphilitic orchitis.**

—(1) Under treatment *resolution* is very common, the function of the gland remaining. (2) The exudation may become indurated, and organise to imperfectly fibrillated connective tissue, but it more frequently undergoes *caseous and mucoid degeneration*, especially when the inflammation is localised in the form of a gumma. Sometimes the degenerated products of a gumma are partially absorbed, and the remainder encapsuled by dense fibrous tissue. After a long duration, the exudation of diffuse orchitis, together with the débris of the secreting structure, may be slowly removed by absorption, leaving the gland atrophied and firm. (3) *Acute suppuration* is rare, chronic much less so. The scrotal tissues become adherent to the parts beneath, an abscess is formed and bursts, then ulceration takes place, and the disintegrated gummatous material escapes with the pus around. In patients who are broken down in health suppuration may be more active and isolate the caseous mass, which is then liberated and cast off as a slough.

**Condition of the tunica vaginalis.**—

Hydrocele is frequent, as might be expected from the body of the gland being so commonly the seat of the disease in question. The *fluid* resembles the

secretion of simple irritative dropsy in other serous membranes. On being drawn it may remain in the liquid state, or form a gelatinous coagulum.

The surface of the visceral layer of the tunica vaginalis is sometimes irregular, from nodules of lymph. Unless there be sufficient fluid to keep the visceral and parietal layers apart, adhesion is very likely to take place between them, so that the cavity is partially or entirely obliterated.

*State of the cord.*—The vessels are dilated, and in long-standing cases their walls are thickened; but the exudation is rarely sufficient to weld them together into a firm indurated mass.

*General considerations.* — Syphilitic sarcocoele, whether diffuse or gummatous, is usually found in the late secondary and tertiary stages of the disease. Both testicles may be affected, but very frequently only one is involved. The deposit rarely begins in the epididymis, and when it does the cord is much more likely to remain free from induration than in strumous testis. As a rule the inflammation is very amenable to specific treatment.

#### **Tubercular or scrofulous sarcocoele.**—

As in scrofulous disease of other tissues, the inflammation here shows itself in the form of diffuse exudation, or as isolated and confluent miliary tubercles or granulations, or as a combination of the two varieties.

Tubercular orchitis and epididymitis is usually found in patients who have a family or personal history of phthisis, or other manifestations of tubercular disease. There is a great liability for both testicles to be affected, though months or years may intervene between the periods of accession of the inflammation in the two organs.

In the majority of cases the disease commences in the globus minor of the epididymis, and for a long time it may be confined to that situation; but sooner

or later it spreads, and the testis itself is involved. Instead of uniform swelling, the outline of the tumour is very irregular. The vas deferens, and it may be the entire spermatic cord, is thickened. On examination per rectum the vesiculæ seminales and the vasa deferentia may be found to be free, or indurated and enlarged (Fig 68c). Tubercular nodules may exist in the prostate. After a time the scrotum becomes congested, œdematous, and adherent to the structures beneath.

The further course is uncertain. Under treatment the inflammation may subside, leaving a nodular hardness in the epididymis, to which the scrotum remains fixed by puckered adhesions. But the tendency is to caseation and chronic abscess. The whole organ may be converted into a cheesy mass without suppuration occurring; but as a rule the degenerated tissue breaks down, and the débris mingles with the exudation from the surrounding vessels. Then fluctuation can be detected, and little by little the integument thins, and finally it bursts, and the contents of the cavity are evacuated.

Before this happens the whole of the secreting structure may be destroyed; but far more commonly the abscess points and opens whilst it is yet localised. The lining wall is covered on the free surface with pus and disintegrating tissue; next to this is a belt of vascular granulation tissue.

From this point the disease may take one of two departures: (1) Filling in of the cavity with lymph that organises and cicatrises—*i.e.* spontaneous cure; (2) progressive suppuration, and destruction of the remaining portion of the gland. The former is the exception. The latter in its progress develops.

**Hernia testis—Fungous testis**, like hernia cerebri, is essentially inflammatory. The mere opening in the tunica albuginea does not explain it;



if it did, this would be sufficient ground alone to contra-indicate the treatment of acute orchitis by puncture. Nevertheless, the resistance offered by the unyielding fibrous capsule is an important factor in the process, for whilst the volume of the contents tends continuously to increase, by reason of the exudation from the vessels, accumulation is prevented by the rigidity of the inexpandible investment of the organ. Hence it follows that so long as exudation exceeds absorption, the surplus must escape through the opening established by the bursting of the abscess.

But it is not simply a filtering of pus through the inflamed tissue, since this would not account for the protrusion. Fibrin separates from the liquor sanguinis and embeds the migratory leucocytes; and into the semi-solid product capillary loops shoot from the adjacent vessels; and thus a vascular granulation tissue is formed which constitutes the greater part of the fungus.

Meanwhile the glandular tubules are broken up; but before their disintegration is complete, columns and isolated cells of epithelium are carried outwards by the advancing granulation tissue with which they are incorporated, and can be recognised with the microscope. The fungus does not attain a large size, for the superficial layer melts away whilst the growth is replenished from beneath. After a time it becomes stationary, and with the subsidence of the inflammation it recedes. The secretion is at first purulent, then muco-purulent, or thin and serous. Its nature and quantity depend upon the degree of inflammation. The skin around is congested, and in long-standing cases the papillæ and hairs are hypertrophied.

There is a milder form of "fungous testis," in which the glandular structure seems to be but little affected. A small abscess forms beneath the scrotum,



and opens externally. This leaves a sinus, terminating in an orifice surrounded by pale gelatinous granulations. On manipulation an indurated cord can be felt, taking the direction of the sinus, and fixed to the



Fig. 64.—Tubercular Testis, showing the Formation of Abscess. The preparation was injected with Gelatin and Prussian-Blue.

- a*, Spermatic cord much enlarged, the vessels are embedded in indurated lymph; *b*, portions of the testicle apparently quite healthy, the tubules are seen presenting a fringed appearance on the surface of the section; *c*, irregular mass of cheesy material, the artificial injection has stopped abruptly at the periphery; *d*, small abscess pointing at the front of the organ. The epididymis, which has lost its outline, forms a solid mass at the upper and back parts of the testis, continuous with the thickened cord. (Reduced one quarter.)

testis or epididymis. It probably begins as an isolated tubercular deposit in the epididymis.

Hernia testis may result from the breaking down of a syphilitic gumma. It is more often situated on the anterior part of the scrotum than tubercular hernia

testis, as would be expected from the seat of election of the inflammation in the two diseases.

**Section of a tubercular testis.**—The appearance on section will depend upon the rate at which the structure has been undermined, and the time at which the examination is made. In comparatively acute cases the whole organ may be broken down into a semi-diffuent pulp, suppuration having completed the destruction begun and continued by gradual obliteration of the vessels and consequent fatty degeneration.

But the testis undergoes considerable enlargement, and caseation is for the most part well advanced before the occurrence of suppuration. Large tracts are slowly deprived of their vascular supply, and the constituent elements break up into molecular fat. Meanwhile a gradual desiccation takes place, with the result that the disorganised tissue and exudation are left as irregular, firm cheesy masses, which appear granular or quite smooth on the cut surface. Artificial injection shows these masses to be non-vascular, and makes them stand out in marked contrast to the surrounding tissue, which is coloured by the fluid that finds its way into the, as yet, patent vessels.

Fig. 64 represents a strumous testis removed from a man æt. 30. The other gland had been excised three and a half years before for the same disease. The cord is thickened. A portion of the secreting structure appears healthy. There is a yellowish-white caseous mass at the centre and to one side. Suppuration has commenced and already destroyed the investing tunics, and caused the skin to bulge. During life fluctuation could be detected at this spot.

**Minute anatomy of tubercular sarcocele.**

—It was formerly supposed that the disease began as an inflammatory effusion *into* the tubuli seminiferi, and that as the walls of the latter became softened

and distended, they ruptured and discharged their contents into the intertubular spaces. But this is not the case. The essential pathology is a *chronic interstitial inflammation* (Fig. 20). The connective tissue is increased by infiltration with liquor sanguinis and leucocytes. This may be very decided before there is any change in the epithelial lining of the tubes. After a time, however, the cells become granular, and, as they proliferate, are set free in the lumen, which eventually becomes choked with desquamative and exudative products. The morbid processes can be studied better in the epididymis than in the testis proper, for in it the tubes are much wider. The blood-vessels get blocked by coagula and compressed by exudation. The result of this is a starvation of the tissues they supply, and, as a natural consequence, fatty degeneration and caseation. Or the interruption in nutrition may be more acute as the effusion from the vessels is more rapid and copious. Then, instead of caseating, the neoplasia liquefies, and suppuration is established. On the other hand, the lymph may organise into a low form of cicatricial tissue. Bearing these facts in mind, we can understand the diversified appearances in this group of cases.

**Disseminated tubercular orchitis.**— This variety of strumous testis is generally seen in the course of a more widely-spread miliary tuberculosis, particularly through the genito-urinary tract. Even when the disease appears to be localised in the testis there is great liability to its subsequent outbreak in other organs. I have known tubercular meningitis follow disseminated tubercle of the testis in the adult. Fig. 68 depicts the bladder of a patient who had tubercular ulcers of the urethra, and in whom the kidneys, prostate, vesiculæ seminales, and vasa deferentia were affected with caseating miliary granulations. But it must be remembered that the diffuse tubercular

epididymitis before described is also prone to be followed by the formation of miliary tubercles in the same organ, or in distant parts.

Anatomically the disease is characterised by the development of grey granulations between the tubules of the epididymis and testis. At first they are discrete, but subsequently they become more or less confluent by their own growth; or by the intercalation of fresh tubercles; or a number of granulations may be hidden in the exudation products of inflammation between and around them. They soon undergo fatty degeneration and caseation, and as they soften in the centre small abscesses are formed; by the aggregation and fusion of these abscesses irregular cavities with festooned margins are made.

Miliary tuberculosis of the testis is more common than is generally supposed, as the patients frequently die from the invasion of the more vital organs before the local lesion is sufficiently advanced to be recognised during life. Both testicles are usually involved, and the vasa deferentia and vesiculæ seminales appreciably thickened (Fig. 68).

**Malignant sarcocoele.**—Malignant disease of the testicle includes encephaloid cancer, sarcoma, and chondro-sarcoma. Colloid cancer is very rare.

The features that serve to distinguish new growths involving the testicle from inflammatory enlargements are as follow :—

1. *Size.* This may be quite decisive, for whilst a tumour may exceed in size the fœtal head at full term, simple, syphilitic, and strumous sarcocoele rarely attain a greater magnitude than a goose's egg.

2. *Lobulation.* Malignant growths are not uncommonly lobulated, not so much on the surface as in their internal structure, for the resistance offered by the tunica albuginea serves to diffuse the pressure and maintain the even outline of the mass. The

caseous patches of tubercular testis and syphilitic gummata are irregular in shape, and contrast strongly with the surrounding tissue.

3. Smooth-walled *cysts* are characteristic of new growth or simple cystic dilatation. They cannot be mistaken for the cavities formed by softening and suppuration in tubercular disease.

4. Malignant tumours that are very rapid in their development, especially encephaloid cancer, often appear flocculent on the cut surface when the preparation is placed in fluid. Inflammatory deposits, on the other hand, are generally quite smooth on section.

5. As malignant growths are plentifully supplied with blood-vessels, *injections of coloured fluid* penetrate the entire mass. The yellow caseous tracts of tubercular and syphilitic disease are extravascular, and consequently the injection stops short at their periphery.

Whilst one or more of the above-mentioned criteria may fail in a given case, the evidence afforded by their collective consideration can never leave a doubt as to the nature of the enlargement.

**Encephaloid cancer** is usually met with between the ages of twenty-five and forty-five. It commences in a proliferation of the epithelium of the seminiferous tubes. The growth is generally very rapid. The whole of the secreting structure is destroyed before the tunica albuginea gives way. When this takes place the scrotal tissues are quickly involved, and the skin, at first simply adherent, gets deeply congested, and finally it ulcerates, and the unrestrained growth protrudes as a bleeding mass. "*fungus hæmatodes*."

The uniform elasticity of the tumour goes a long way in differentiating it from tubercular and syphilitic lesions. On puncture, blood escapes quite freely.

sometimes bringing with it minute portions of growth, the structure of which can be recognised under the microscope.

Cysts are much less frequent than in sarcoma.

The spermatic cord is always enlarged by dilatation of the blood-vessels to meet the wants of the

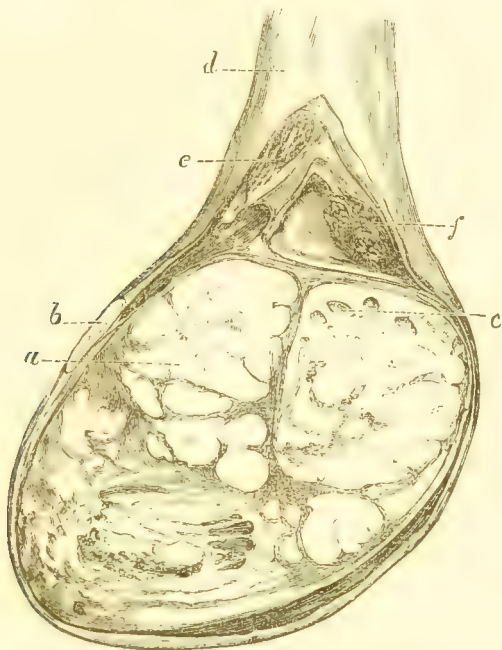


Fig. 65.—Chondro-sarcoma of the Testicle.

The normal structure has entirely disappeared. The new growth, *a*, presents a regular lobulated appearance. At *c* several small smooth walled cysts are seen. With the exception of the nodule *b* the cartilage is disposed in microscopical islets in the substance of the softer sarcomatous base; *f*, caseous debris of inflammatory exudation in upper part of tunica vaginalis; *e*, blood-vessels filled with artificial injection; *d*, spermatic cord enlarged, but free from invasion by the growth. (Reduced one quarter.)

rapidly-growing mass. It may also be indurated by the deposit of cancer in the lymphatics; but this I have found to be the exception rather than the rule.

If the glands are affected, they will be found to



be the lumbar in connection with the testicle. As the integument becomes involved, those in the groin may suffer as well.

Except in very rare cases, the disease attacks only one testicle. For an account of the naked-eye and microscopical characters, *vide* Encephaloid cancer (page 581).

**Sarcoma** occurs at the same period of life as cancer, but it is also met with in children under ten years of age. It begins in the connective tissue of the gland or in the tunica albuginea. I have known it attain a large size without invading the testicle at all, the gland substance being spread out over the tumour, and this is not very unusual. In clinical gravity it vies with cancer. It has a great tendency to generalise in the internal organs. The cord and lymphatic glands are sometimes infiltrated.

It is very frequently cystic. Its minute structure varies between round, mixed, and spindle cells. Cartilaginous transformation is not rare (Fig. 65).

**Enchondroma** is rather a chondrifying sarcoma than a primary cartilage growth. The greater portion of the neoplasia may be converted into cartilage. In such a case the term enchondroma is admissible on anatomical grounds, but its affinities with sarcoma should never be lost sight of. The cartilage may exist in the form of microscopical islets or in large sinuous tracts or clumps. On hardening these tumours in spirit the cartilage contracts less than the surrounding soft sarcomatous tissue, and thus the cut surface is rendered uneven. Secondary growths possess the characters of the primary.

The cartilage is mostly of the hyaline variety.

In these tumours there is sometimes a considerable amount of gelatinous or mucoid tissue.

**Cystic sarcocoele.**—It would appear that some cases of cystic testicle are developed from constriction

and dilatation of the seminiferous tubes of the rete testis and the body of the organ. The large majority are, however, not of this nature, but are dependent upon mucoid or colloid degeneration of the cells and intercellular substance of some pre-existing solid growth.

The presence of a number of cysts in a tumour of the testis at once raises the presumption of its sarcomatous structure, for experience shows that here, as in all other situations, cystic sarcoma is of much more frequent occurrence than cystic cancer.

The entire mass may be riddled with small cysts, many of which are of microscopical dimensions, embedded in a soft greyish-white ground substance; or the cysts may be of large size, and so numerous as to give a honeycombed appearance on section. The walls of the cysts are for the most part quite smooth, and the contents clear, and either pale or stained with the colouring matter of the blood from minute capillary hæmorrhages. Other cavities or accidental cysts owe their origin to interstitial extravasations and fatty softening from deficient vascular supply.

Secondary intracystic growths sometimes partially or entirely fill the spaces. In the latter event the nature of affairs is manifest from the absence of structural continuity, except at the base of attachment, between the proliferating buds and the inner surface of the walls of the cysts.

When the tumour is of slow growth the neoplasia passes to a higher phase of organisation, so that the intercystic trabeculae appear quite fibrous.

## CHAPTER LXVI.

## ATROPHY OF THE TESTICLE.

SMALLNESS of a testicle does not necessarily imply atrophy. It may be a case of imperfect development. Undescended testicles are below the normal size, whether they occupy the inguinal canal or remain in the abdomen free from unusual pressure. It is true the resistance offered by the walls of the canal may hinder the growth of the organ, but defective development is rather the cause than the consequence of arrested descent.

The causes of atrophy are (1) inflammatory lesions ; (2) excessive functional activity ; (3) senile decay ; (4) long-standing varicocele, by preventing the renewal of a proper amount of nutritive fluid, and by the continuous pressure of the distended vessels upon the tubules ; atrophy from this source never amounts to impotence (Paget) ; (5) blocking of the vas deferens.

## CHAPTER LXVII.

## HYDROCELE.

THE term hydrocele is applied to, for the most part, watery or serous exudations into (1) the tunica vaginalis testis; (2) the whole or some part of the funicular portion of the processus vaginalis peritonei; (3) dilated tubes of the epididymis or rete testis, or the hydatid of Morgagni, or the vas aberrans of Haller; (4) loculi in the spermatic cord, which are said to correspond to free cystic formations in the cellular tissue of other parts. Possibly some cases are developed in connection with the organ of Giralvés.

**Hydrocele of the tunica vaginalis.**—So far as one can tell, this often constitutes a disease *per se*, i.e. it arises without any precedent morbid condition of the testicle. In such cases it is generally found at the two extremes of life—infancy and old age. In the former period it seems to originate in the locking-up of peritoneal fluid in the tunica vaginalis, as this is shut off from the funicular process by the physiological closure of the canal at the top of the testicle. The tension upon the membrane probably stimulates it to further secretion, for simple withdrawal of the contents is usually sufficient to effect a cure.

The hydrocele of old people is said to be the result of degenerative changes in the tunica vaginalis, which shows itself in a “loss of balance between secretion and absorption.” It is due, then, either to hypersecretion, or to obstruction of the lymph vessels that naturally carry off the fluid from the serous sac; or

it may to some extent be compensatory to the atrophy of the testicle incidental to advanced age, in the same way that fat fills up the spaces of wasting bone and serous effusion takes the place of a shrinking brain.

The *nature of the fluid* is primarily that of the normal secretion of serous membranes, though it is frequently modified by secondary changes consequent on increased irritation, and more rarely by the accidental rupture of blood-vessels into the sac. As a rule, it is quite clear and straw-coloured. It contains a considerable amount of fibrinogen. It is albuminous, as shown by coagulation by heat or nitric acid. Occasionally it holds in suspension numerous crystals of cholesterine, the product of fatty metamorphosis. These may be so plentiful as to cause the fluid to sparkle when it is viewed by transmitted light. Granules and crystals of hæmatoidin are of rarer occurrence; they are the permanent evidence of previous effusion of blood.

*Condition of the sac.*—The walls are usually thin enough to allow of translucency of the swelling. In some cases they are much thickened and indurated, especially after repeated tapplings. The inner surface is smooth, or exceptionally irregular from chronic inflammatory exudation.

Vaginal hydrocele is often secondary to disease of the testicle, especially orchitis (chap. lxiv.).

The measures taken to obtain a cure of simple chronic hydrocele aim at obliteration of the cavity by inflammatory adhesion, or by such alteration of the secreting surface as entails a cessation of the abnormal effusion. An analogous process is that set up by injection of iodine into joints the seat of hydrops or chronic serous synovitis.

In all cases of acute inflammatory hydrocele the fluid is highly fibrinous.

## ENCYSTED HYDROCELE OF THE EPIDIDYMIS.

This is most commonly the result of obstruction and subsequent dilatation of one of the vasa efferentia. Now and then it consists of an enlargement of the vas aberrans (Fig. 67). It may be simulated by distension of the corpus Morgagni, or of the organ of Giralde's, or encysted hydrocele of the contiguous part of the

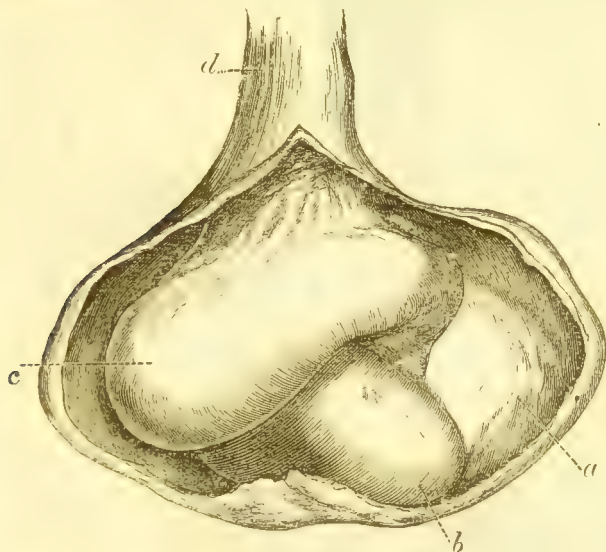


Fig. 66.—Encysted Hydrocele of Epididymis.

*a*, Cavity of tunica vaginalis ; *b*, testicle ; *c*, hydrocele ; *d*, spermatic cord  
(Reduced one quarter.)

spermatic cord. In the first instance it is situated above the testicle, but as it increases in size it invaginates the tunica vaginalis, the cavity of which it may entirely fill (Fig. 66).

*The contents* are either clear and serous or semi-opaque and opalescent, like water rendered slightly turbid from admixture with milk. In the latter case spermatic filaments are present in abundance, and the tumour is known as "*spermatocele*." According to



Curling, the fluid is of this nature from the commencement, or what was originally limpid has become cloudy

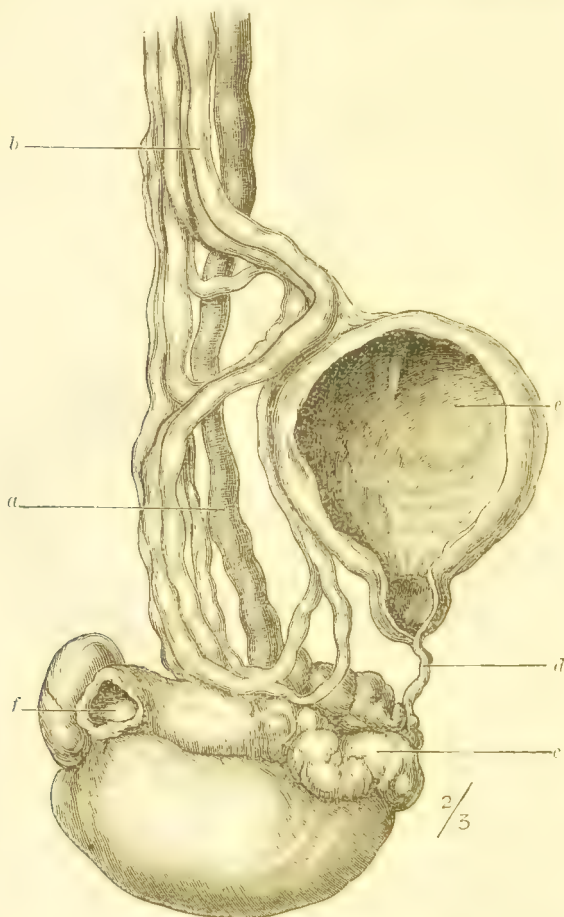


Fig. 67.—Encysted Hydrocele of Epididymis.

*a*, Vas deferens; *b*, spermatic veins; *c*, epididymis; *d*, vas aberrans; *e*, cyst of the vas aberrans; *f*, cyst of globus major, so-called encysted hydrocele of tunica vaginalis. (St. Mary's Hospital Museum.)

from rupture of a seminal tubule into the sac of the hydrocele.

## CONGENITAL HYDROCELE.

Here the process of peritoneum brought down with the descent of the testicle remains patent throughout, and the passage opens into the abdominal cavity by a smaller or larger orifice. Reduction of the fluid is usually quite easy, but some part of the tract may be so narrowed that continued pressure is required to effect it. Should the orifice be closed the case is one of *infantile hydrocele*.

## HYDROCELE OF THE CORD.

*Hydrocele of the cord* is either encysted or diffuse. Encysted hydrocele is due to incomplete closure of the funicular process of the peritoneum. When very tense, and occupying the inguinal canal, it may be mistaken for an enlarged gland.

*Diffuse funicular hydrocele* is reducible or irreducible. In the former case the continuity of the funicular and vaginal sections of the serous tube is broken by adhesion of the opposed surfaces of the membrane above the testicle, but the communication with the general peritoneal cavity is maintained. In the irreducible variety this also is closed, so that there is an isolated sac co-extensive with the whole or greater part of the cord. Instead of one space, there may be a series of encysted hydroceles, giving a moniliform appearance. Some authors describe another form of diffuse hydrocele, in which the fluid is said to distend the hollows of the areolar meshwork of the cord; a local œdema, in fact. The cause of this is difficult to understand, for there are none of the other signs of inflammation present, and simple mechanical congestive exudation would probably be associated with varicocele, a condition by no means constant. In the female the canal of Nuck may be the seat of collections of fluid corresponding with

those met with in the funicular process of the peritoneum in the male.

#### HÆMATOCELE.

Hæmatocele, or a collection of blood in the cavity of the tunica vaginalis, may be caused (1) by a blow on the testis, (2) by puncture of a vein in tapping a hydrocele, (3) by the sudden diminution of tension in the latter operation.

## CHAPTER LXVIII.

## GONORRHOEA AND ITS CONSEQUENCES.

**General pathology.**—Some pathologists believe that gonorrhœa is a disease due to the presence of specific micrococci, whilst others deny this, and maintain that it is merely a purulent urethritis capable of being set up by other conditions than impure intercourse. Abuse of alcohol and excessive sexual indulgence are deemed sufficient for its causation. Certain vaginal discharges, concerning which there is no reason to suppose a specific origin, not infrequently excite acute and subacute catarrhal inflammation; such *e.g.* are the products of leucorrhœa and menstruation. The fact that the disease is contagious does not prove that it is specific, for the chemical bodies formed in unhealthy inflammations have a cauterant action on the tissues. Moreover, constitutional infection is the chief sign of a specific disease, and in the great majority of cases gonorrhœa is a purely local disorder. Even when constitutional symptoms do manifest themselves, they are not pathognomonic of specific infective inflammation, for simple idiopathic suppuration in other parts is known to cause them in certain subjects.

**Morbid anatomy.**—Gonorrhœa, like acute inflammation of other mucous membranes, first shows itself in increased secretion of mucus, *i.e.* in unusual nutritive and functional activity of the epithelial cells. But soon the exudation of leucocytes becomes very active, and the migratory cells mingle with embryonic corpuscles derived from segmentation and endogenous multiplication of the epithelium. Pus is freely poured out. The submucous tissue is infiltrated with liquor sanguinis and cells, and the membrane in its

entire thickness is swollen. There is considerable induration, and the corpus spongiosum may feel quite firm and cord-like. The vessels are set as it were in a bed of plastic matter, which prevents them from expanding equally with those of the corpora cavernosa; hence the chordee. In very acute cases the tension on the capillaries is so great that occasionally some of them burst, and blood escapes from the urethra.

As the inflammation subsides, the discharge gets more watery. With proper treatment it disappears altogether, for the natural termination is in cure. Not one case in a hundred ends in chronic thickening and stricture. Nevertheless, the part remains a locus resistentiæ minoris, and subsequent attacks are very easily excited, though not usually with the virulence of the primary one.

**Consequences of gonorrhœa.**—These are local and general, immediate and remote. The immediate local consequences include cystitis, prostatitis, epididymitis, bubo, urethral abscess, and retention of urine. The remote local consequences are stricture and its results, cystitis, epididymitis, surgical kidney, hypertrophy of the bladder and ureters, extravasation of urine, urinary abscess, etc. The term "local" is here used to imply the structures in anatomical relationship with the urethra. The general consequences are dependent on absorption of some irritating matter from the seat of primary inflammation. In very rare cases acute fatal pyæmia is induced; more often the symptoms are less severe, and then the constitutional state is designated "gonorrhœal rheumatism," on account of the prevalence with which articulations and fibrous investments are involved, rather than on any known character of the virus showing its analogy with the materies morbi of idiopathic rheumatism. Gonorrhœal ophthalmia

occurs as a generalised affection, and as an accident from direct inoculation.

**Gonorrhœal cystitis.**—Gonorrhœa is the most common cause of cystitis. It is due to spreading of the inflammation in the continuity of the mucous membrane, and not to regurgitation of pus. It is possible that catheterism may be exceptionally the means of conveying the discharge. The cystitis is most marked about the neck of the bladder. It is recognised symptomatically by pain and frequency of micturition; and anatomically by swelling and mucopurulent catarrh of the mucous membrane. When gonorrhœa ends in pyæmia, it is generally by destructive cystitis, for in these cases the mucous and submucous tissues are infiltrated with pus, and the membrane may be hæmorrhagic and sloughy. On the supposition that gonorrhœa is not a specific disease, we must say that pyæmia is not due solely to the virulence of the products of the primary non-infective inflammation, but to a true infective process grafted on to the original simple urethritis.

**Gonorrhœal epididymitis.** — (*Vide* Acute orchitis, pages 394–396.)

**Retention of urine.**—This is partly the result of the static obstruction offered by the swollen mucous membrane, and partly of the dynamic resistance due to spastic contraction of the sphincter vesicæ. Reflex paralysis of the expelling muscles of the bladder will not account for it, since the most powerful voluntary contractions of the abdominal muscles are unable to overcome the difficulty. The intolerable suffering of the patient contrasts with his passive endurance of retention from enlarged prostate and long-standing stricture. This is explained by the excessively sensitive condition of the inflamed structures, and by the fact that the bladder is not accustomed to acute distension of its walls; another exemplification of the



law, that morbid states suddenly induced show themselves much more by signs and symptoms than those of slow development. It is generally associated with acute prostatitis.

**Gonorrhœal stricture of the urethra** will be treated of in the general description of organic stricture, since the pathological changes do not materially differ from those concerned in the production of other forms of fibrous contraction, and the remote results are the same.

**Gonorrhœal affections of the eye.**—These are: (1) Gonorrhœal rheumatic scleritis; (2) double catarrhal conjunctivitis; (3) purulent ophthalmia (generally unilateral) due to inoculation.

(1) *Scleritis*.—Although the symptoms are mainly referred to the sclerotic, it must not be supposed that the contiguous structures are not affected; no more than that periostitis exists without a certain amount of osteitis. The physical signs are increased tension of the globe; injection of the sclerotic vessels sometimes localised or intensified at the place of attachment of the external recti muscles; and congestion of the ocular conjunctiva, etc. In persons predisposed to rheumatism, and especially in those of mature or advanced age, the morbid changes and symptoms are those of ordinary rheumatic ophthalmitis. In fact, the diagnosis turns chiefly on a knowledge of the cause.

(2) *Gonorrhœal conjunctivitis* seems to be due to absorption of some irritating substance from the urethra, and its diffusion in the general circulation; for not only are both eyes affected, but the symptoms are liable to recur with recurrence of the urethritis. It can scarcely be a reflex nervous disorder, for there is neither physiological nor anatomical connection between the structures primarily and secondarily involved. The inflammation is shown by hyperæmia and mucous or muco-purulent discharge.

(3) *Purulent ophthalmia* is mostly, if not always, caused by direct inoculation. It is one of the most destructive diseases of the eye. In many cases the sight is lost, and in most it is permanently damaged. As a rule only one eye is affected. The palpebral conjunctiva is greatly swollen, and the ocular overlaps the edge of the cornea (chemosis). The cornea inflames and then ulcerates, the ulceration usually taking place at the periphery, for the pressure is greater there than at the centre on account of the chemosed conjunctiva. Sometimes it sloughs, owing to the stoppage of the circulation in the plasmatic canals. The interlaminar spaces are crowded with pus cells (onyx). In the worst cases the eye-ball is destroyed by suppuration and sloughing, only a shrunken caruncular mass remaining; short of this the cornea remains opaque, and perhaps bulged (anterior staphyloma), or it is collapsed owing to escape of the vitreous humour.

## CHAPTER LXIX.

## STRICTURE OF THE URETHRA.

THREE kinds are generally described : (1) Organic ; (2) congestive ; (3) spasmodic.

**Spasmodic stricture.**—Authors differ as to the frequency and degree of functional, dynamic, or spasmodic stricture. There seems no reason to doubt its actual occurrence, for there is the anatomical basis in the extrinsic and intrinsic muscles of the urethra ; and, moreover, a catheter may be grasped and tightly held during its passage at one time, whilst it slips into the bladder with great readiness at another. It may constitute a disease *per se*, or, rather, it may be the only symptom of undue nervous or muscular excitability ; more often it complicates congestive and organic strictures.

**Congestive stricture** is seen to perfection in retention from acute gonorrhœa. It adds to the difficulty of micturition caused by organic stricture and enlarged prostate, and so explains those cases where urine can be voided one day whilst it is retained the next. The irritation that causes it may be mechanical, chemical, or functional, and it may act directly upon the part, or at a distance. The passage of a catheter constitutes a mechanical irritant ; excessive use of alcohol, and other local and general stimulants, a physico-chemical irritant ; and forced retention of urine, or sexual excitement, a functional irritant. Exposure to wet and cold evidently acts in a reflex manner.

The *modus operandi* probably varies in different cases. The possible explanations are : (1) Paralysis of the muscular fibre cells of the vessels, or terminals of

the nerves, from immediate injury ; (2) reflex vaso-constrictor paralysis ; (3) reflex vaso-dilator stimulation.

Passive congestion of long-standing is at once the cause and consequence of organic stricture, for the irritation maintained by the stricture keeps the blood-vessels dilated ; and this is accompanied by more or less exudation, induration, and contraction, like nutmeg cirrhosis of the liver from passive hyperæmia of the hepatic veins. After a time the vessels lose their tone, and are unable to contract, and this may remain long after the primary source of irritation has been removed.

**Organic stricture.**—Gonorrhœal urethritis is by far the most common cause of organic stricture ; but traumatism is not rare. The urethra may be injured from without by blows upon the perineum, or from within by forcible instrumentation, or impaction of a calculus.

In any case the natural elastic tissues of the urethra are substituted by inflammatory exudation which undergoes cicatricial contraction. In the majority of instances the entire circle of the urethra is involved, and the stricture is consequently annular. This is the necessary result of complete rupture. Localised ulceration from the pressure of an impacted calculus, although it leads to a certain amount of contraction, may cause but little difficulty, as a considerable portion of the circumference of the walls remains intact.

The canal of the urethra is only a potential one, for when the parts are at rest the opposite surfaces are in contact. The expansion of the walls under the pressure of the column of urine during micturition is more than counterbalanced by the contraction of the newly-formed connective tissue ; and so great is the resistance offered by the stricture, that the powerful exertion of a hypertrophied bladder is unable to cope

with it beyond the extent of keeping the passage permeable.

Post mortem a stricture does not appear so tight as during life, for all vital contractility is abolished, and softening of the fibrous elements sets in soon after death.

The so-called "resiliency" of a stricture, which accounts for recontraction within a short period of mechanical dilatation, is probably due to combined elasticity and contractility. Whilst there are few or no muscular fibres in the stricture itself, there is reason to believe that the difficulty experienced in catheterism is partly owing to the spastic contraction of the muscles outside. This view is supported by the fact, that although a catheter may be tightly gripped soon after engaging an organic stricture, if the parts be allowed a short period of rest the instrument afterwards travels with comparative ease. Of course the blood-vessels are relieved at the same time, but their previous fulness does not seem sufficient to explain the difference in the degree of resistance.

A **bridle stricture** is one where fibrous bands stretch from one part of the urethral wall to another. It is possible that in some cases these trabeculæ are formed by the making of false passages through the base of the stricture; but as a rule they are of natural construction. Absorption of the central or external portions (short of the entire length) of membranous projections into the canal of the urethra would leave the free edges as the cords in question; the same as occurs physiologically in the development of the chordæ tendineæ of the heart.

The **effects of treatment**.—An organic stricture is never cured, for whatever treatment is adopted the patient cannot dispense with the occasional use of a bougie.

The different methods employed aim at one or

more of three attainments: (1) Stretching of the cicatricial tissue; (2) absorption of inflammatory products; (3) the interposition of new plastic matter (splicing). In the process of stretching by "gradual dilatation" the tissue elements are elongated, and in all probability dislocated from one another at the same time. In "forcible dilatation" this is undoubtedly the case; in fact, the mechanism may be described as multiple interstitial laceration, for it is doubtful if the stricture ever gives way in one place alone. Absorption of inflammatory products, which exist in the form of recent exudation, and partially or perfectly organised fibrous tissue, is effected by the pressure of a catheter, especially when this is allowed to remain in the stricture for some time. The blood-vessels are mechanically supported, and so endosmosis is favoured, and the result is atrophy from continuous pressure. The process is analogous to the dispersion of chronic inflammatory thickening of a limb by strapping and bandaging. In internal urethrotomy the stricture is divided, and the edges gape; the latter condition I verified by post-mortem examination in a case fatal soon after operation. Lymph is effused about the wound, and the interval is filled up by plastic matter; the same in kind, but less in degree, as when a tendon is elongated after tenotomy. Catheterism, subsequent to section, stretches the young connective tissue, and with it, though to a slighter extent, the material of which the stricture was originally composed.

When there is much induration outside the urethra, internal urethrotomy may fail to divide the entire thickness of the stricture. It is in these cases that external urethrotomy is of such signal service.



## CHAPTER LXX.

URINARY ABSCESS—EXTRAVASATION OF URINE—  
URINARY FISTULA.

**Urinary abscess** is usually preceded by stricture, which is in fact its principal cause. The tension upon the urethra at and behind the stricture sets up inflammation, and this leads to ulceration through the walls, and consequent escape of a small quantity of urine under high pressure; or the inflammation spreads to the cellular tissue outside the urethra, and there ends in suppuration. In the latter case a communication between the urethra and the abscess cavity is subsequently established. It is not always easy to tell the order of sequence, but from the fact that the cavities of some perineal abscesses due to stricture are entirely free from the channel of the urethra, and that occasionally urine first makes its appearance at the wound some time after the abscess has burst, or has been opened, it seems clear that the second mode of formation is by no means infrequent. Whatever may be the exact mode of formation, a barrier of lymph is thrown out around the abscess, and this checks the sudden outrush of urine and extravasation into the spaces of the areolar meshwork. The pressure of the inflammatory exudation outside the urethra adds to the difficulty of the urine escaping by the natural passage, and to the chances of its finding its way into the abscess. In this way the tension becomes so great that unless it is relieved by free incision there is great danger of the wall of the abscess breaking down, with consequent extravasation.

The obstruction to the flow of urine may be

lessened by ulceration and sloughing of the strictured part of the urethra. In very rare cases the relief thus afforded is sufficient to provide for the discharge of the contents of the abscess per urethram. As a rule, however, an artificial opening is made in the perineum, and through this the pent-up matter escapes. Subsequently the cavity of the abscess shrinks, but its complete closure is often prevented by the pressure of the urine behind the stricture, and *fistula* is the result.

The contents of the abscess consist of pus, urine, and the débris of tissue. They are very offensive from putrefactive decomposition.

The pressure of an impacted calculus may cause an opening in the urethral wall by ulceration, and in this way the calculus may escape into the sub-urethral tissues, and there set up suppuration. The pus from such an abscess may make its exit entirely by the urethra, since with the disappearance of the calculus from the natural passage the chief source of obstruction to the flow of urine is removed. Such cases prove that high tension is a far more potent cause of urinary abscess and extravasation than the chemical irritation of the tissues by urine. So long as the calculus remains in its new position it is subject to increase in size, from the deposit of urinary salts as the fluid bathes its surface, for it is very seldom that the orifice, by which the cavity lodging the stone communicates with the urethra, closes. The incrustation of the calculus may lead to difficulty in micturition later, in consequence of encroachment upon the urethral channel.

Urinary abscess in connection with the penile portion of the urethra is usually the result of impacted calculus.

**Extravasation of urine.**—Either the bladder or the urethra may give way. When the former ruptures

through its serous covering acute peritonitis is set up. When the rent occurs below the attachment of the false ligaments pelvic cellulitis is the consequence. The causes of urethral extravasation are stricture, blows in the perineum, fracture of the pubic bone, and impacted calculus. In children the last-mentioned cause is the most common.

“Extravasation” differs from “urinary abscess” in that the urine is rapidly diffused through the interstices of the cellular tissue, instead of being limited to a comparatively small space. This depends (1) upon the size of the opening in the urethra, (2) upon the degree of obstruction in its lumen. The loose areolar tissue in the perineum and scrotum offers but little resistance to the escape of urine, as the latter is forced along under high pressure. The irritative nature of the fluid and the great tension upon the blood-vessels soon lead to stasis and widespread thrombosis and gangrene. The parts become enormously swollen. At first they are tense and red, then they become boggy and discoloured; for with the cessation of the circulation exudation is checked; and the hæmoglobin of the blood is decomposed. The derivative pigments variously tinted cause the skin to assume a mottled and variegated aspect—red, green, black, etc. Subcutaneous emphysematous crackling can be felt. This is due to the evolution of gases of putrefaction. On cutting into the gangrenous tissues a sero-sanguinolent fluid escapes. If suppuration have preceded the general extravasation the discharge will contain much pus. The local treatment is directed to two ends:—(1) Relief of inflammatory tension, and escape of decomposing fluids; (2) establishment and maintenance of a free passage from the bladder; or, in other words, the removal of the effects of past extravasation, and the prevention of further mischief. So long as the

lymphatics and blood-vessels remain exposed to putrescent matter the danger of septic absorption continues. Asthenia and septicæmia are the consequences to be feared and guarded against.

The path of the extravasation is directed by the attachments of the deep membranous layer of the superficial fascia ; hence it passes from the perineum to the scrotum, and so on to the abdomen. Limited yet fatal extravasation has been known to follow tapping of the bladder through the rectum and above the pubes.

**Urinary fistula.**—The bladder may communicate with the rectum or vagina, and the urethra with the external surface, rectum, or vagina. *Rectovesical* fistula is generally the consequence of some operative measure, such as rectal lithotomy, or tapping of the bladder per rectum ; but it also arises during the progress of a pelvic abscess. In the latter case a direct passage is opened up between the two viscera by ulceration or sloughing of their contiguous walls ; or a sinuous tract intervenes. *Vesico-vaginal* fistula results from long-continued pressure of the foetal head during parturition, and from vaginal lithotomy. Ordinary perineal urinary fistula is a common sequel of urinary abscess and extravasation. These unnatural passages remain open from one or both of two causes : obstruction to the flow of urine *per viam naturalem* ; and want of rest, which entails the absence of those conditions necessary to the union of two opposed granulating surfaces. No amount of treatment is likely to be successful in a case of perineal fistula whilst a stricture of the urethra remains undilated, for there is a frequently recurring distension of the walls of the fistula by the stream of urine diverted from its proper course. The physiological contraction of the muscles of the bladder, vagina, and rectum causes one granulating surface to

glide upon the other, and so keeps up continual irritation, and exudation from the vessels. An important point in the management of these cases after operation is, as far as possible, to relieve the viscera involved of their functional activity.

Where fistulous tracts are long and sinuous, it is often necessary to lay them freely open, otherwise the external openings may contract, or even close, before the granulations in the deeper recesses cease to secrete more than suffices for their agglutination. By this unequal contraction, cul-de-sacs, or segments of the main sinus, become filled with pus, which goes on accumulating until the tension is high enough to burst open the original orifice, or a gradual process of ulceration establishes a new one.

## CHAPTER LXXI.

## HYPERTROPHY OF THE BLADDER.

HYPERTROPHY of the muscular coat of the bladder is the result of increased functional activity. As a rare event this happens in children from excess of intrinsic nervous or muscular irritability of the organ. With this exception hypertrophy may be considered as consequent on obstruction to the flow of urine in some part of the urethra. Enlargement of the prostate and organic stricture are by far the most common antecedents. In these cases the hypertrophy is purposive or compensatory. It is called into existence by the stimulus of increased resistance (Fig. 7).

Up to a certain extent, hypertrophy is able to keep pace with the obstruction ; but, as a rule, sooner or later the pressure becomes so great that dilatation is superadded, the same as in "dilated hypertrophy" of the heart (Fig. 70). The walls of the bladder may be so expanded that the hypertrophy is masked. The true index of its existence is the amount or bulk of the entire muscular coat, and not simply its thickness. When the distension is very great, there is comparative powerlessness, and the condition is known as *atony*. *Ceteris paribus*, the atony is proportionate to the acuteness of the distension. There are several factors concerned in its production : (1) Exhaustion of muscular irritability from repeated futile efforts to overcome the resistance ; (2) deficiency of innervation ; (3) degeneration of the muscular fibre cells.

As long as the bladder is able to empty itself, the obstruction may be very great without causing atony ; for, in the intervals of physiological rest, repair takes place. Permanent tension means exhaustion. A



consideration of these facts explains why, in most cases, dilatation succeeds hypertrophy from enlarged prostate, and why, as an exceptional circumstance, the hypertrophied bladder remains empty and contracted, and is smaller than the prostate.

The difficulty interposed in the way of free circulation through the vessels of the mucous membrane leads to atrophy of the more highly developed elements and areolar hyperplasia of the submucous tissue.

**Hypertrophy and dilatation of the ureters.**—*Vide* Surgical Kidney, pages 452–56. The pathology and morbid anatomy are the same as in the corresponding states of the bladder.

## CHAPTER LXXII.

## CYSTITIS—ULCERATION OF THE BLADDER.

THE *causes of cystitis* are (1) spreading of the inflammatory process from one of the neighbouring passages, as in gonorrhœal urethritis; (2) tension on the walls of the bladder, as in retention from stricture, enlarged prostate, and fracture of the spine; (3) irritation of the mucous membrane by the chemical products of decomposition of urine; (4) mechanical irritation from a calculus, etc.; (5) vaso-motor and trophic changes consequent on injury or disease of the central nervous system; (6) new growths, tubercular infection; (7) certain drugs, *e.g.* cantharides and turpentine.

It frequently happens that more than one cause is concerned in producing cystitis; *e.g.* in fracture of the spine there is tension from the retention of urine; perverted nutrition of the mucous membrane from disordered innervation; acute congestion from paralysis of the vaso-motor nerves; and sometimes, though unwarrantably, septic decomposition of urine from the passage of an unclean catheter. Decomposition of urine is both a cause and consequence of cystitis.

In the viscid mucus of chronic catarrh there exists a ferment capable of setting up the alkaline fermentation. In consequence of this, urea is converted into carbonate of ammonia. But this is not the only product of the decomposition, and, probably, not the chief one, in so far as the cause or increase of the inflammation is concerned.

**Varieties of cystitis.**—These may be arranged on two bases: (1) As to the intensity of the inflammation, acute and chronic; (2) as to the cause, *e.g.* calculous cystitis, tubercular cystitis, etc.

The term "catarrh" is somewhat misleading ; for clinically it refers to chronic and subacute inflammation, whilst pathologically it receives its interpretation more or less in every case of cystitis.

**Acute cystitis** consists of increased multiplication, mucoid transformation, and shedding of the epithelial cells, and of exudation of liquor sanguinis and migration of leucocytes. The *products* vary in appearance and consistence according to the degree of inflammation. At first they are comparatively thin, the serum of the blood mingling with and diluting the mucus derived from dissolution of the distended epithelial cells. A number of corpuscles are held in suspension, but not as yet in sufficient numbers to cause more than a cloudiness of the fluid. As the inflammation heightens, the discharge becomes mucopurulent and then purulent ; but in every stage it contains a considerable quantity of mucin. Then, as resolution takes place, it diminishes in quantity, and gets more glutinous.

In comparatively rare cases the exudation is highly fibrinous, coagulating upon the surface of the mucous membrane, and forming a cast of the interior of the bladder. Anatomically, it resembles the false membrane of croup and the casts of plastic bronchitis.

When the inflammatory congestion is very intense, capillary hæmorrhages occur both in the interstices of the membrane and on its surface ; the exudation is consequently more or less sanguinolent.

Stasis and thrombosis may be so extensive as to entail death of portions of the mucous membrane. Flakes or shreds of slough are set free by liquefaction of their attachments, and floated off by the stream of exudation and the urine with which they are bathed.

The fluid passed per urethram varies not only as

the characters of the inflammatory products, but as the amount of urinary admixture and the existence and degree of decomposition. Remaining but a short time in the bladder, it is usually acid.

**Chronic and subacute cystitis** are exceedingly common as the result of hypertrophied prostate and stricture of the urethra. In both these diseases there is difficulty in completely emptying the bladder. Very often it amounts to impossibility, so that after each effort at micturition there is a residuum of urine. This is very liable to decompose, and increase and perpetuate the cystitis.

When the urine is allowed to stand, it will be found that thick ropy mucus clings so tenaciously to the bottom of the vessel that inversion of the latter does not suffice to disengage it. It is frequently alkaline in reaction when passed; if not, it very quickly becomes so. It has an offensive ammoniacal smell. Besides mucus, it contains pus and dirty grumous matter, consisting of epithelial cells, blood corpuscles, and amorphous debris. It deposits triple phosphate.

The mucous membrane is deeply congested, much swollen, and sometimes pigmented to a marked degree. The summits of the temporary rugæ, and even broader tracts, are often encrusted with sabulous material (inspissated mucus and pus impregnated with earthy salts).

The ureters and kidneys are generally diseased.

Cystitis varies from acute purulent infiltration and discharge to chronic mucous catarrh. The terms "acute," "subacute," and "chronic" do not denote fixed pathological landmarks. They are used to indicate groups of symptoms and morbid appearances of comparative but indeterminate intensity.

There may be a certain amount of cystitis in the acute fevers and other pyrexial conditions.

**Ulceration of the bladder** is caused by (1) the mechanical irritation of a stone, etc. ; (2) the breaking down of tubercular deposits ; (3) malignant disease ; (4) destructive inflammation arising from other



Fig. 68.—Tubercular Ulceration of the Bladder.

*a*, Miliary granulations ; *b*, excavated ulcers ; *c*, vesicula seminalis ; *d*, vas deferens, enlarged from tubercular inflammation. (Reduced one half.)

sources ; *e.g.* gonorrhœa, septic infection of a lithotomy wound, etc.

Ulceration from the pressure and friction of a stone is generally situated about the neck of the bladder, since this is the part most subject to injury when the viscus empties itself. The ulcer is comparatively superficial, and without marked induration

of the base. It is usually associated with advanced cystitis of the entire mucous membrane.

Tubercular ulceration is due to the softening of miliary tubercles and consecutive destruction of the surrounding tissues. The ulcers are multiple, as a rule (Fig. 68). Though most common at the base, they are often indefinitely distributed. Their margins are sharply defined, and frequently undermined. By the coalescence of contiguous ulcers the mucous and submucous tissues may be destroyed over a wide expanse. Tubercular ulcers of the bladder, ureters, and urethra may be found in the same subject, together with miliary granulations in the kidneys, testicles, and other organs. Bacilli can be demonstrated in the urine.

Malignant tumours of the bladder ulcerate early, for in addition to their intrinsic tendency to disintegration, they are subject to injury by muscular contraction.

When treating of acute cystitis it was remarked that the circulation might be so far arrested as to lead to ulceration and sloughing of the mucous membrane. This is all the more likely to occur where the inflammation is infective.



## CHAPTER LXXIII.

## TUMOURS OF THE BLADDER.

TUMOURS of the bladder, like those of other hollow viscera, are liable to assume the polypoid or villous form. This is notably the case with regard to benign growths, but the malignant ones are not exempt. The peculiarity depends more upon unequal resistance than on a special predisposition of the new formation to enlarge in a certain direction. On account of the varying amount of support, consequent on the state of tension of the walls of the bladder, and the disturbance of the circulation in the tumour caused by contraction of the muscular coat, hæmorrhage is usually a prominent symptom. It is more likely to occur, and in profusion, in those cases where the blood-vessels are large, numerous, thin-walled, and embedded in soft loose tissue; *e.g.* in simple villous tumour and villous cancer. Bleeding may be the first, and for a long time the only, indication of a growth. But the diagnosis may often be verified by a microscopical examination of the urine, and especially that drawn off by a catheter. In one case I found numerous delicate tufts of villous growth, and in another a minute fragment of columnar epithelioma. This method of investigation is of great importance where the existence of a benign tumour is suspected; since there is an absence of induration of the walls of the bladder.

**Malignant tumours of the bladder** comprise villous cancer, epithelioma, and sarcoma. They are mostly primary, beginning in the bladder or neighbouring parts, vagina, rectum, or prostate.

*Villous cancer* is very rapid in its growth. The

surface of the tumour is flocculent from delicate outgrowths and shreds of disintegrating tissue. The base is broad, and not limited to the mucous membrane. In fact, there is infiltration of the walls of the bladder. The trigone is the usual seat of the disease. The surrounding structures may be involved. Hæmorrhage and painful and frequent micturition are the chief symptoms.

*Epithelioma.*—Authors differ as to the relative frequency of epithelioma and encephaloid cancer of the bladder. There are two facts that help to explain the discrepancy; firstly, cases of sarcoma have been included with soft cancers; and secondly, both the tumours in question (epithelioma and encephaloid cancer) are of epithelial origin, and the disposition and relative amount of stroma and cells vary. The natural surface epithelium of the bladder is flattened, and the cells of the deeper layers are fusiform or columnar. Each variety may be represented in the new growth. I have met with pure columnar epithelioma. The tumour is of firmer consistence than encephaloid cancer, and the surface is generally smoother, it may be unevenly lobulated.

*Sarcoma.*—The clinical and pathological characters are those of round and spindle-celled sarcoma generally.

**Benign tumours** of the bladder are almost invariably polypoid, or villous. They have a special tendency to grow from the trigone near the orifices of the ureters. By far the most common variety is the simple *villous tumour* (Fig. 92). This may be single or multiple. Occasionally it is diffuse, the greater part of the mucous membrane being covered with a shaggy, flocculent coat. The villi are arborescent in some cases, filiform in others. The mucous membrane appears healthy close up to the insertion of the outgrowths, or only unusually rough from fine

villosities. There is no invasion of the deeper structures. This is also true of the other benign growths. It is an important feature, since it serves to distinguish "villous tumour" from "villous cancer" even in the early stages of development of the latter,

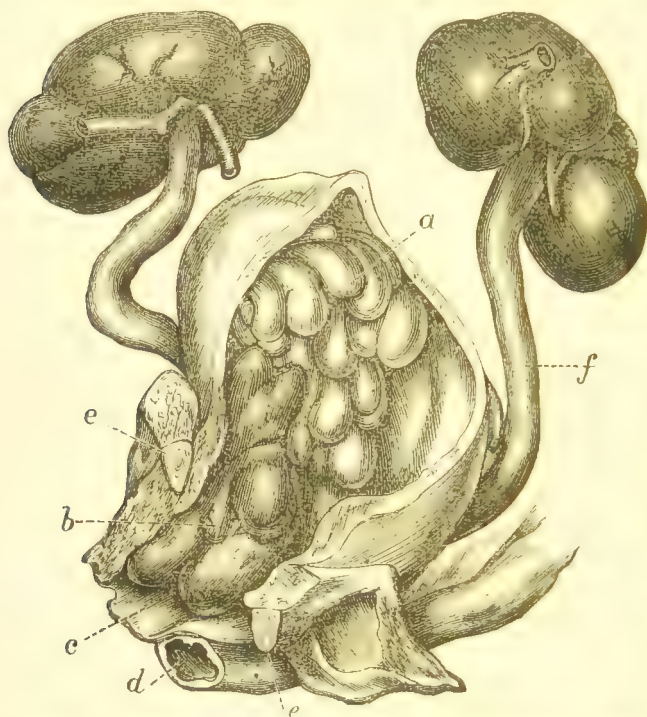


Fig. 69.—Mucous Polypi of the Bladder of a Female Child, aged eighteen months.

The majority are attached around the orifice of the right ureter. The urinary organs have been removed in their entirety, together with the rectum and a portion of the pelvis. *a*, Polypi; *b*, the same prolapsed, and congested from partial strangulation by the dilated urethra; *c*, vagina; *d*, rectum; *e*, cut surface of divided symphysis pubis; *f*, dilated ureter. (Reduced one half.)

when the two formations have such a close surface resemblance to one another. The basis of the villous or papillary processes consists of a delicate stroma of fibrous or mucous tissue richly supplied with large

capillary vessels. The epithelium may be squamous, or columnar, and disposed in few or many layers. Granules of blood pigment, scattered, or in groups, may frequently be seen in the substance of the growth. These are the remnants of interstitial extravasations, and not the result of true physiological pigmentation by the agency of the protoplasm of the cells.

Next in order of frequency to villous tumour comes fibroma, or *fibrous polypus*. It is distinctly pedunculated, and of firm, fleshy consistence.

*Mucous or gelatinous polypus* is a great rarity. Paget records two cases. Figs. 69 and 82 were taken from one that came under my notice at University College Hospital. With the exception that the epithelium is squamous, these polypi differ in no essential particular from the common nasal variety.

A pedunculated outgrowth from the prostate sometimes projects into the bladder. Clinically it may be classed with the other vesical tumours, but pathologically it is quite distinct from them.

## CHAPTER LXXIV.

## HÆMATURIA.

BLOOD may escape from any part of the urinary tract, from the Malpighian glomeruli of the kidney down to the spongy portion of the urethra. In the diagnosis of its source one has to pay attention, amongst other points, to its quantity, colour, degree of admixture with the urine, and the period at which it makes its appearance with regard to the act of micturition.

When the blood comes from the kidney it is usually quite dark, from reduction of the oxyhæmoglobin by the urine; but there are exceptions to the rule. I have known it to be florid in copious bleeding from a cancerous tumour. Very large effusions generally come from the kidney or the bladder. Profuse hæmorrhage from the urethra is known by the fact of its escaping independently of the act of micturition.

**Hæmorrhage from the kidney** may be in small or dangerously large quantity. The causes of severe hæmorrhage are (1) malignant tumour; (2) laceration of the kidney; (3) renal calculus; (4) congestion in acute Bright's disease; (5) reflex hyperæmia, from irritation of some part of the urinary passages—*e.g.* by the dilatation of a stricture of the urethra; (6) disturbance of the spinal nervous system. (*Vide* page 159.) Tubercular disease of the kidneys and other morbid states occasionally give rise to it.

The bleeding from primary cancer and sarcoma of the kidney may be very profuse, but its occurrence is by no means certain. One cause of immunity is

obstruction of the ureter by a projection from the growth. Secondary cancer is less vascular than primary, and it usually exists in the form of nodules, instead of a soft fungoid mass, so the chances of hæmorrhage are more remote.

In laceration of the kidney the hæmaturia is likely to be very great, unless the organ is completely crushed, or the ureter ruptured.

In acute Bright's disease the bleeding is salutary within a certain range. It is nature's mode of giving relief to the over-distended vessels. The mechanism consists of high blood-pressure and diminished power of resistance from degeneration of the walls of the capillaries.

The hæmorrhage from reflex hyperæmia can be explained in two ways: (1) a general flooding of all the vessels of the kidney, from vaso-motor paralysis; (2) contraction of the renal arteries, and, as a consequence, back pressure from the veins.

**Hæmorrhage from the ureters** is never very great. The causes are (1) the passage of a calculus; (2) simple and tubercular inflammation.

**Hæmorrhage from the bladder**, when profuse, is strongly suggestive of villous growth, or acute ulceration opening up a comparatively large vessel. *Intermittent hæmaturia* depends on the presence of an animal parasite, *Bilharzia hæmatobia*. It may extend over many years. Adults rarely get rid of the disease.

Other causes of vesical hæmaturia are calculus and cystitis.

**Hæmorrhage from the prostate and urethra.**—Prostatitis, prostatic abscess, malignant disease, and catheterism should be inquired after in the former case. Traumatism from without and within, over-distension of the erectile tissue of the corpus spongiosum, acute urethritis, urethral chancre,



and the passage of a calculus, in the latter. In old men with enlarged prostates hæmorrhage from the veins and capillaries is at times sudden and copious. It must be borne in mind that "blood in the urine" is not synonymous with "hæmaturia." The admixture may take place after the urine is voided, the blood being derived from some other source than the urinary tract.

**Hæmoglobinuria.**—In this condition the blood-corpuscles are broken up and the colouring matter is diffused through the urine. (*Vide* page 361.)

## CHAPTER LXXV.

## DISEASES OF THE PROSTATE GLAND.

**Hypertrophy of the prostate** is a disease incidental to advanced age. The morbid anatomy is sufficiently precise, but the ætiology is unknown. The entire gland may be the seat of diffuse hyperplasia, or a portion of it only may be enlarged, or circumscribed nodules may form in its substance (the so-called "prostatic glandular tumours"); or, lastly, similar masses may project from its surface, as in the analogous case of the thyroid. What were formerly designated "fibrous tumours" of the prostate are now known to consist in the main of unstriped muscular fibre-cells. Histologically, nothing is observed that is not typified in the normal structure (Fig. 71).

The nearest approach is made by "uterine fibroids," but the latter do not contain glandular elements. Hypertrophy of the prostate stands, as it were, on the borderland of new formations or growths and chronic inflammatory neoplasia; but the general likeness does not imply intrinsic pathological affinity.

So long as the prostatic urethra is not encroached upon, the gland may assume considerable proportions without giving rise to symptoms.

**Cancer of the prostate** is not common. The encephaloid variety is said to be that usually met with. The few cases that I have seen were hard enough to merit the name "scirrhus." The symptoms generally are those of stone in the bladder. At first the induration and enlargement simulate the signs of chronic prostatitis and prostatic hypertrophy. Later, the acuteness of the patient's suffering, the wasting of the body, the repeated hæmorrhages, and, it may

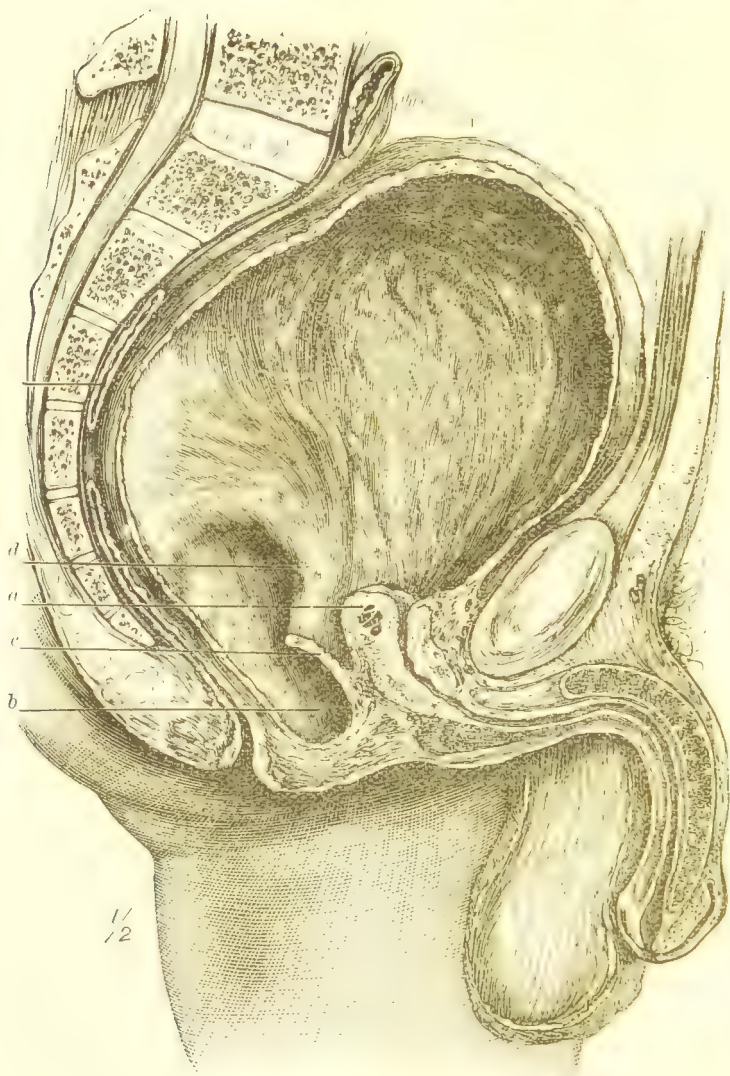


Fig. 70.—Enormously dilated Bladder from an old Man.

*a*, Hypertrophy of middle lobe of prostate, which contains a cystic space; *b*, pouch of bladder behind prostate corresponding with trigone; *c*, ridge formed by wall of bladder at level of urethral orifices; *d*, orifice of left ureter. The enlarged middle lobe of the prostate acted like a valve, and so obstructed the urethral opening. The pouch *b*, which completely overlaps the prostate, formed a receptacle for retained urine. It will be seen with what difficulty a calculus lying in the lower part of the pouch would be struck by a sound. (St. Mary's Hospital Museum.)

be, the implication of surrounding structures, clear up the diagnosis.

**Prostatitis and prostatic abscess.**—Acute prostatitis is mostly a sequel of gonorrhœal urethritis,



Fig. 71.—Section of a so-called Prostatic Glandular Tumour (Lobulated Hypertrophy).

A nodule as large as a walnut was shelled out during the extraction of a vesical calculus. *a*, Glandular recess; *b*, involuntary muscular fibre; *c*, blood-vessel.  $\times 265$ .

but it may be caused by instrumentation, impaction of a calculus, and other modes of injury. The gland is excessively tender. It is swollen and indurated. Pus may escape from the inflamed prostatic urethra

without the occurrence of parenchymatous suppuration. Prostatic abscess is recognised by throbbing pain, and by the detection of bogginess or fluctuation per rectum, and by the discharge of matter in considerable quantity. It usually bursts into the urethra, but it sometimes opens into the rectum, or in the perineum. Piles occasionally develop meanwhile. Inflammation and pus formation may occur around the gland (periprostatitis).

Chronic prostatitis is the sequel of an acute attack, or the inflammation is of moderate degree from the first. Gleet, syphilis, vesical calculus, and catheterism are the chief causes. The morbid changes are the same in kind as in the acute disease. The gland may be left permanently hard and enlarged. On section it looks greyish and somewhat translucent, not unlike a moderately firm scirrhus tumour. The secreting glands and ducts are marked by pale-yellow spots and streaks.

**Tubercle of the prostate** is seldom seen except in conjunction with a similar affection of the epididymis, vasa deferentia, and vesiculæ seminales. The kidneys, ureters, bladder, and urethra may be involved at the same time.

The tubercles are at first discrete, then they become confluent. The tubercles and the exudation products in which they are embedded caseate, and the mass sometimes breaks down, forming an abscess with purulent and cheesy contents, as in a strumous lymphatic gland.

Tubercle of the prostate is more common than is generally supposed. In all cases of tuberculosis of other parts the prostate should be examined.

**Corpora amylacea** are said to be usually present in the prostates of adults. They are of microscopic dimensions, or of such a size as to be clearly visible to the naked eye. Individually they may



attain a diameter of from  $\frac{1}{12}$  to  $\frac{1}{6}$  of an inch. Collectively they sometimes form masses of considerable magnitude, being embedded in some cementing substance, and enclosed in a common capsule. They are for the most part composed of concentric laminæ. In their interior may be seen granular particles, or even nuclei and cells. They may be coloured brown or black. The chief alteration to which they are subject is calcification. They have been found in phleboliths in the prostatic veins. (*Vide* page 150.)

**Prostatic calculi.**—Those of intrinsic prostatic origin probably begin as a deposit of organic matter in the glandular acini or ducts. This would be followed by calcareous infiltration and accretion.

The constituent salts are phosphate and carbonate of lime, chiefly the former.

These calculi may be single or multiple. By their pressure the tissue of the prostate is more or less absorbed.

They may be encapsuled, or lie loose in a cavity, or project into the prostatic urethra.

When there are several in the same subject they are liable to be faceted by mutual attrition; and as felt per rectum they may give a grating sensation as they are made to rub one against the other.

A vesical calculus may become impacted in the prostatic urethra, and ultimately embedded in the substance of the gland.



## CHAPTER LXXVI.

## SURGICAL KIDNEY.

SURGICAL kidney consists essentially of an interstitial or intertubular nephritis. It differs from granular or gouty kidney as follows: (1) The inflammatory changes are more irregularly disposed; (2) the progress of the disease may be very rapid; if chronic, it is subject to repeated exacerbations; (3) it frequently ends in suppuration; (4) it is usually accompanied by cystitis and pyelitis; (5) the concomitant and consequent organic changes are limited to the urinary apparatus; there is no hypertrophy of the heart, nor a general systemic arterio-capillary fibrosis.

**Causes.**—These come under two heads: (1) Those that raise the tension in the renal tubes and capillaries; (2) septic poisoning.

The tension may be increased by interference with the escape of urine from stricture of the urethra; enlarged prostate; obstruction of one or both ureters, from (a) impacted calculus, (b) stricture from ulceration and compression by a tumour, or organised lymph from pelvic cellulitis;\* and paralysis of the bladder in fracture of the spine.

Although the urine does not regurgitate from the bladder into the ureters, the result is the same as if it did; for if the bladder is distended, it is clear the urine must be dammed back upon the ureters and renal tubules. The distension of the tubules compresses the vessels that encircle them, and so causes a congestion of the capillaries on the distal side of the obstruction. Again, the tension may be raised from *reflex irritation* of some part of the urinary tract, by a stone in the bladder, or operations upon

\* *Lancet*, vol. i. p. 769; 1879.

the urethra ; or the vaso-motor paralysis may be of central origin, as in crushing of the spinal cord.

*Septic poisoning* may take place : (1) By spreading of the inflammation in the continuity of the mucous membrane from the bladder, ureter, or pelvis of the kidney ; (2) by lymphatic absorption from these parts.

Dickinson accounts for the nephritis, and the scattered foci of the inflammation, by embolism of the renal veins ; but the limitation of the infarctions to the kidney, and their shape and local distribution, seem opposed to the theory of embolic pyæmia.

Johnson's explanation of the dissemination of the abscesses and patches of inflammation is that they are due to rupture of the renal tubules ; but if this were correct, suppuration should be more constant than it is. My own view is that primary thromboses occur here and there in the obstructed intertubular capillaries (cortical and medullary), and that localised perivascular inflammation occurs about them.

Nephritis following injury to the spinal cord may be due in some measure to trophic lesions of the kidney.

**General anatomy.**—The appearance of the kidney varies, according to the cause and acuteness of the inflammation.

In **chronic interstitial nephritis, from obstruction** to the flow of urine, the effects of atrophy from pressure are combined with those of inflammation. The papillæ are flattened or entirely absorbed, their places being occupied by recesses continuous with the calyces. In extreme cases the pyramids may disappear, and only a thin layer of cortical substance remain.

If the disease be limited to one kidney, every vestige of secreting substance may vanish, and the capsule may then retain its natural outline, or be converted into a large thin-walled cyst. I have known

such mistaken during life for an ovarian tumour. Atrophy from pressure is more pronounced when the disease is unilateral, for then the other kidney hypertrophies, and compensates for the impaired function of the crippled organ, which is thus relieved from its physiological work.

The effects of inflammation are: (1) Induration from organisation and contraction of the exudation; (2) an irregular nodulation or puckering of the surface from cicatrisation; (3) adhesion of the kidney to the capsule, and the capsule to the perirenal fat; (4) cysts beneath the capsule and in the cortical substance from obstruction of the secreting tubes.

**Acute interstitial nephritis.** — Here the kidney is paler and softer than normal, being infiltrated with leucocytes. It is spotted and streaked with dilated vessels and minute extravasations both in the cortex and pyramids. The small-celled infiltration may be uniform, but more often it is unequally distributed, and then there are pale nodules, and these in different stages of softening and abscess formation. The abscesses are usually multiple, but the greater part of the kidney may be occupied by one large abscess formed by the coalescence of smaller ones. The capsule is adherent, but can be readily separated. There may be suppuration beneath the capsule, and in rare cases outside it. Sometimes there are streaks of suppuration within the pyramids. The inflammation is probably septic in all cases.

**Acute supervening on chronic inflammation.** — The appearances will consist of a mixture of the signs of the above-mentioned types.

**Condition of the ureters and pelvis.** — Much will depend upon the cause, duration, and intensity of the disease. *When due to chronic obstruction* they are dilated, and their walls thickened from inflammatory exudation and muscular hypertrophy.

They are often strictured from contraction following inflammation or cicatrisation of ulcers. The mucous membrane is thickened and hyperæmic.

In *acute cases* the passage contains a mixture of pus and decomposed urine, often tinged with blood from capillary hæmorrhages. They rarely contain clear acid urine. The mucous membrane is swollen

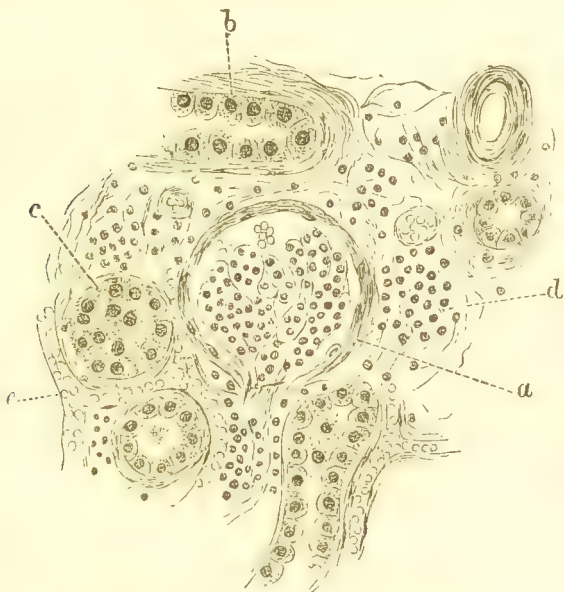


Fig. 72.—Early stage of Interstitial Nephritis (Surgical Kidney), from a case of Polypus of the Bladder, in which the Orifices of the Ureters were obstructed. (*Vide* Fig. 69.)

*a*, Glomerulus, the vessels are hidden by corpuscles stained with logwood; *b*, renal tubule; *c*, tubule containing epithelial debris and desquamated cells; *d*, intertubular tissue increased and infiltrated with leucocytes; *e*, capillaries.

and deeply congested, and it may show points of capillary extravasation and slate-grey patches of degenerating lymph.

**Microscopy.**—(1) *Chronic inflammation.*—In the early stages there is congestion and exudation of leucocytes, mostly about the Malpighian bodies, the

capsules of which are slightly swollen and homogeneous. The tubules are somewhat dilated (Fig. 72). The epithelium is but little altered; at the most the cells are flattened, or show signs of commencing proliferation and cloudy swelling.

In advanced cases there are wide tracts of cicatricial tissue, in which but few vessels and no tubules can be seen. In other parts the disease is more active, there being a rich infiltration with cells, and increased structural change in the secreting epithelium; but even yet some portions may appear fairly healthy.

(2) *Acute inflammation.*—The whole organ may be in a state of diffuse inflammation, the interstitial tissue being everywhere crowded with cells. The tubules contain altered epithelium and casts of fibrin, and even of leucocytes.

But the disease is usually concentrated upon certain points where nothing can be seen but groups of cells, or abscesses in existence or in course of formation. There is a zone of hyperæmia around these foci, and in some parts capillary extravasation as well.

**State of the urine.**—In chronic cases the quantity is increased from the high vascular tension. It may be double the normal. It is pale, and of low specific gravity. At first it contains but little or no albumin. The secretion of urea is diminished.

In *acute cases* the amount of urine may still be greatly above the normal. In one instance, supposed during life to be diabetes insipidus, I found post mortem both kidneys riddled with abscesses. But often the kidney is so crippled that the amount of water is diminished, and there is a daily decrease in the secretion of urea. When cystitis and pyelitis are present the urine contains pus and mucus, and sometimes blood.

The patients usually succumb to a combination of septicæmia and uræmia.

## CHAPTER LXXVII.

## SUPPRESSION OF URINE—URETHRAL FEVER—URINARY DEPOSITS AND CALCULI.

**Suppression of urine** is obstructive or non-obstructive. In the former case it generally follows impaction of a calculus in one ureter, the kidney on the other side having previously been incapacitated by disease. The condition of the patient is peculiar, for whilst the suppression may last a week or more, the urinous odour of the breath is usually absent, and there is no dropsy.

Death from non-obstructive suppression has followed the passage of a catheter within a few hours. The pathology of such cases is somewhat complex. It cannot be explained by simple reflex congestion of the kidney, for the organ may not be flooded with blood to the extent that one would suppose necessary to account for the total abrogation of function. It may be that the nerve irritation affects the secreting cells directly. The suddenness of onset and the absence of a large wounded surface preclude the assumption of acute septic intoxication being the cause ; but probably all the above-mentioned factors combine in their action. In the majority of cases there is a certain amount of interstitial nephritis.

**Urethral fever** is the name given to the general state caused by some injury to the urethra, such as internal urethrotomy, or forcible dilatation, or even simple catheterism. It is frequently ushered in by a rigor which may be repeated. The temperature rises rapidly. The other symptoms of fever are present. Then there is sweating, and with it a quick decline in the body-heat. Whilst the fever lasts but little urine is passed ; with its disappearance there is often a copious flow.



Sometimes a considerable amount of blood is lost, and this seems to come from the kidney, for it is well mixed with the urine, unless in such quantity as to coagulate in the bladder—a circumstance which has happened twice in the author's practice (once after Holt's dilatation and once after easy catheterism)—and its effusion is attended with decided relief from pain in the back. The cause of the fever is apparently fourfold: (1) Reflex congestion and consequent impairment of function of the kidney; (2) stimulation of the nerves of an unusually sensitive tract; (3) absorption of poisonous matter, though this must be very slight in cases where but little or no injury is done to the urethra; (4) nervous irritability of the patient.

I have known it ensue more than once in the same case upon the withdrawal of a catheter which had lain in the bladder for 48 hours without causing the least disturbance.

In most, if not in all, cases the kidney has been previously damaged by inflammatory changes, especially those found in interstitial nephritis (surgical kidney).

TABLE OF URINARY DEPOSITS.

NAME.	CHARACTERS.	CAUSES.	SYMPTOMS.
Urates or Lithates of Ammonia and Soda.	Pinkish-yellow, red, or lateritious (brick-dust) sediment; urine scanty, acid, and high-coloured. The precipitate, before subsiding, forms a cloud in the urine, which clears off when heated.	1. Rapid waste of tissues, <i>e.g.</i> as in fevers; 2, excess in nitrogenous food; 3, dyspepsia; 4, obstructed perspiration; 5, congestion of the kidneys (Golding Bird). Also imperfect respiration. Cold weather will precipitate urates sometimes from healthy urine.	Those of the causes. Sometimes also a slight burning feel in passing water.
	Crystalline form—uric acid, mostly rhombic prisms and plates. "Gravel."		
Urates. —Minute spheres with acicular spiculae of uric acid projecting from them.			



Fig. 73.—Urinary Crystal

1 Acicular crystals of tyrosin and globules of leucin ; 2, common forms of uric acid crystals ; 3, octahedra-envelope crystals and dumb-bells of oxalate of lime ; 4, fern-leaf crystal of triple phosphate ; 5, common forms of triple phosphate crystals ; 6, hexagonal crystals of cystin ; 7, neutral calcic phosphate ; 8, diabetic sugar ; 9 and 10, amorphous granular urates, and hedgehog crystals of urate of soda.

NAME.	CHARACTERS.	CAUSES.	SYMPTOMS.
Oxalate of Lime.	Crystalline forms: 1, quadrate octahedra; 2, dumb-bell crystals.	"Nervous exhaustion;" dyspepsia; over-work; mental distress; excess of saccharine food or alcoholic liquors.	Those of the causes. Occasionally, loss of sexual vigour, or disorder of the sexual functions.
Phosphates	1. <i>Phosphate of Lime</i> .—White, cloudy mass. Crystals: spherules, dumb-bells, rosettes, oblique hexagonal prisms. 2. <i>Phosphate of Ammonia and Magnesia (triple phosphate)</i> . Crystals (large): triangular, truncated prisms, four-sided prisms, irregular six-sided plates; stellate crystals when ammonia has been added.	Alkaline urine is the immediate cause. It is caused by injuries and diseases of the bladder, especially paralysis and catarrhal inflammations; renal inflammation; spinal injury or disease. Nervous exhaustion; excessive use of alkalis; the alkalinity of the urine is said to result from the metamorphosis of urea into carbonate of ammonia.	Urine is offensive, and often contains mucus. Signs of causative disease.
Carbonate.	Small and delicate crystalline spherules. Drum-sticks.	The causes which determine the change of urea into carbonate of ammonia.	No special symptoms known. Deposit rare.
Blood.	Urine a dirty-red colour; after standing, a slightly flocculent, brownish sediment. Heat coagulates the albumen. There may be blood enough to form a clot; then the urine is dark brownish-red. Or the blood may be quite unmixed with the urine.	1. Kidney disease. Calculi, congestion, inflammation, injury, scurvy, the <i>Bilharzia capensis</i> . Malaria may cause intermittent hæmaturia. Blood from the kidney is generally mixed uniformly with the urine, and forms blood casts. 2. Bladder affections; injuries, stone, tumours. Blood from bladder often flows pure after the urine. 3. Urethra: blood pure, and comes before or with urine, or without urine at all.	Those of cause. Use Heller's test for blood. Heat urine then add KHO and heat again. The phosphates then fall down with the colouring matter of the blood. The sediment has a dirty-red colour by reflection, and a splendid blood-red colour by transmitted light.

NAME.	CHARACTERS.	CAUSES.	SYMPTOMS.
Pus.	Pus-corpuscles, under the microscope, are spheroidal and granular. The pus generally subsides as a dense layer of a "pale greenish cream-colour," which can be mixed thoroughly with the urine by shaking. Not affected by acetic acid. Forms a translucent jelly when liquor potassæ is added. The urine is albuminous.	Abscess, ulceration, or merely catarrh of any part of the urinary passages. 1. Pus from the kidneys is usually diffused throughout urine passed. 2. Pus from bladder is mostly mixed with mucus. 3. Pus from an abscess is usually variable in quantity, and not equally diffused.	Those of the cause.
Epithelium.	Epithelial cells lining urinary passages. See works on general <i>Anatomy</i> . Often in form of casts.	Kidney disease. Ulceration or catarrh of bladder.	Those of cause.

TABLE OF CALCULI.

NAME.	PHYSICAL CHARACTERS, ETC.	CHEMICAL CHARACTERS.
Lithate of Ammonia.	Occurs rarely except in children. Grey, smooth, dusty, non-laminated appearance.	Soluble in boiling water. Add HCl to solution and you get a precipitate of uric acid. Heat with potassium carbonate: ammonia escapes. Blow-pipe burns it away.
Uric Acid.	Smooth or warty. Yellowish or brownish. Concentric structure.	Gives off no ammonia when heated with KHO. Evaporate to dryness with nitric acid. Cool, and add a little $\text{NH}_3$ ; the characteristic deep purple-red murexide is then obtained. Blow-pipe burns uric acid away.
Oxalate of Lime.	Rough, warty, "mulberry" appearance. Very hard. Dark "blood-stained."	Easily soluble in nitric acid. Boil long in a solution of potassium bicarbonate, neutralise carefully with nitric acid; then white precipitates can be formed with solutions of lime, lead, or silver. Blow-pipe reduces it, first to calcium carbonate, then to quick-lime. Heat on platinum foil and it chars. Then add $\text{HNO}_3$ , and it effervesces.

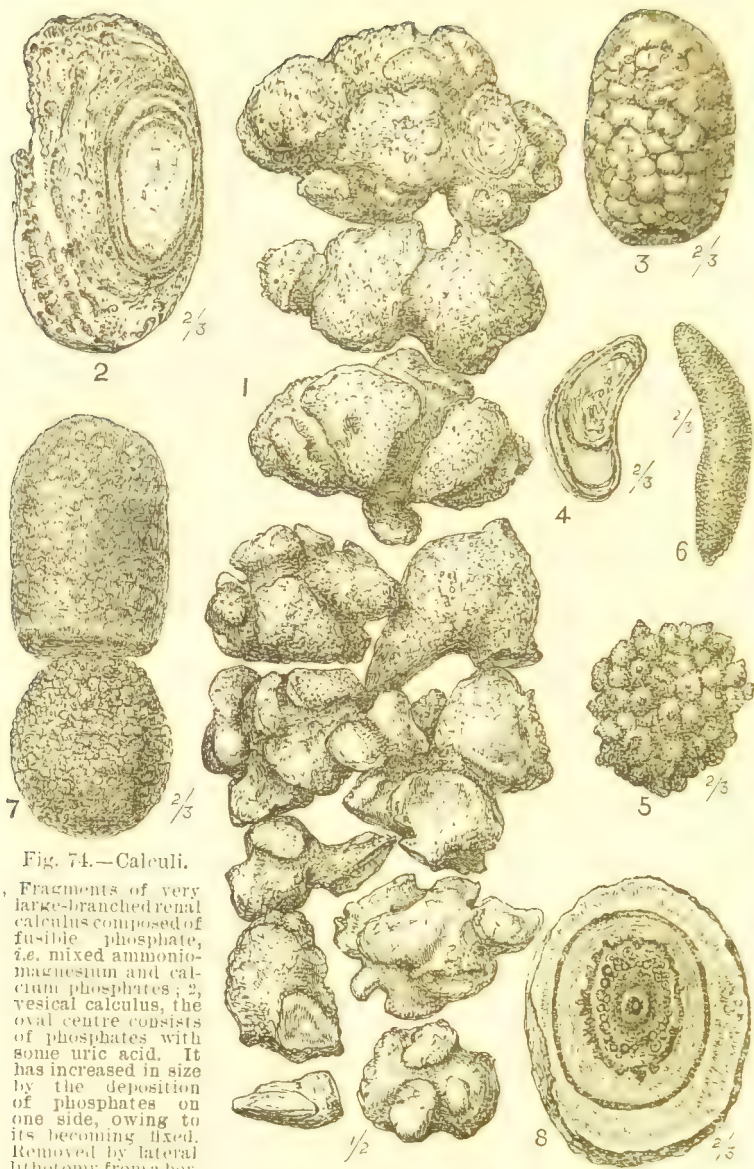


Fig. 74.—Calculi.

- 1, Fragments of very large-branched renal calculus composed of fusible phosphate, *i.e.* mixed ammonio-magnesium and calcium phosphates; 2, vesical calculus, the oval centre consists of phosphates with some uric acid. It has increased in size by the deposition of phosphates on one side, owing to its becoming fixed. Removed by lateral lithotomy from a boy aged 11 years. 3 and 7, cholesterol biliary calculi showing terminal facets; 4, intestinal concretion composed of magnesium phosphate with organic base. It was successfully removed by operation twelve months after an attack of perforative appendicitis. 5, oxalate of lime calculus. The lithotrite failed to crush it. 6, salivary calculus composed of phosphate of lime; 8, alternating vesical calculus; the central part of the stone and the dark narrow zone which divides the lighter part consists of oxalate of lime; the lighter part was shown by analysis to be composed chiefly of oxalate and phosphate of lime. No. 1 is drawn half the real size, and the others two-thirds. (St. Mary's Hospital Museum.)



NAME.	PHYSICAL CHARACTERS, ETC.	CHEMICAL CHARACTERS.
Cystic Oxide.	Has a wavy appearance, especially when fractured. Changes colour with age from pale yellow to brown, grey, or green. Extremely rare. Contains sulphur.	Dissolves, in great part, in ammonia: its solution then deposits, by spontaneous evaporation, six-sided prismatic and tubular crystals. Dissolve in strong caustic potash. Boil, and add a little solution of lead acetate: a black precipitate of sulphide of lead falls.
Xanthic Oxide.	Section, lustrous bright brown. Most extremely rare.	Has a peculiar deep yellow colour, when its solution in nitric acid is evaporated to dryness; characteristic.
Mixed Phosphates of Lime.	Chalky, soft, brittle, laminated.	"Fusible calculus:" melts in the blow-pipe flame. Dissolve in nitric acid and add excess of ammonia: white precipitate.*

**Gall stones compared with urinary calculi.**—(1) Gall-stones are much lighter, the majority, when dry, floating in water; (2) they feel greasy to the touch; (3) the colour of many differs from that of any known form of urinary calculus; (4) the shape is often suggestive, sometimes conclusive, of their nature. The presence of many facets is in favour of biliary origin. If large and barrel-shaped, with terminal facet, there can be no doubt that they are not only gall-stones, but that they were retained for a long time in the gall-bladder, and were multiple. When granular on the surface they look like aggregations of minute calculi, which, in fact, they often are (Fig. 74, Nos. 3 and 7). (5) On fracture, glistening flakes or scales of cholesterine may be very obvious.

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## CHAPTER LXXVIII.

## RENAL CALCULUS—TUBERCULAR KIDNEY—RENAL TUMOURS.

**Renal calculus.**—Calculi originating in the healthy kidney are primarily composed of uric acid or oxalate of lime. The stone, even when large, may be of uniform composition, but not seldom, as the result of pyelitis and nephritis, it becomes encrusted with phosphates. The shape will vary according to the size of the stone and the locality in which it is formed. Thus when small, and situated in the pelvis, it will be ovoid or round; as it enlarges it assumes the configuration of the pelvis, and growing through the calyces it becomes branched; in the latter event the stems of the branches are round, whilst the extremities embedded in the kidney are clubbed or craggy (Fig. 74, No. 1).

Renal calculi, when devoid of phosphatic coating, are often beautifully crystalline on the surface, which then presents a spinous or ribbed appearance. This condition accentuates the pain and increases the liability to hæmaturia.

Uric-acid and oxalate-of-lime stones are first formed in the renal tubules; they may remain *in situ*, or pass into the pelvis and be there retained, or travel into the ureter and become impacted; or lastly, they may reach the bladder and become the nuclei of vesical calculi. Pure phosphatic calculi result from deposit of earthy salts in the pelvis of the kidney as the sequel of pyelitis.

A stone may remain for years in the kidney without causing serious disorganisation, but there is always the danger of suppuration in the organ, in the pelvis, and in the perirenal tissue. The kidney may be converted into a huge abscess, or, its substance having

undergone gradual disintegration, the stone may be found encased by a mass of cicatricial tissue. A calculus permanently impacted in the ureter may lead to suppuration in the kidney or to atrophy of its substance. In the latter event the capsule may be enormously distended with clear fluid.

The prominent symptoms of stone in the kidney are: (1) Pain in the loin, often accompanied by referred pain in the abdominal wall, testis, penis, bladder, and the inner side of the thigh. The intensity and localisation of the pain vary exceedingly; it is generally augmented by movement; occasionally the colic is attended by severe attacks of vomiting. (2) Retraction of the testicle; when present this symptom is of great diagnostic significance. (3) Changes in the urine. The urine at first is often extremely acid and may contain blood-corpuscles; later, profuse hæmaturia may occur. In some cases there is no bleeding. The presence of pus indicates suppuration in the kidney or pelvis; it may be in microscopical quantity, or in such amount as to form a copious deposit.

If the other kidney is healthy the urine retains its acid reaction in spite of the presence of a large admixture with pus, and its specific gravity is not materially altered.

**Tuberculosis of the kidney** may exist as an isolated affection, but more frequently other parts of the genito-urinary system are involved. It may occur in the wake of chronic phthisis, or take part in a general outbreak of miliary tuberculosis. Both kidneys are usually diseased; but the morbid process may for a long time be confined to one organ, and then the simulation of renal calculus may be very marked. It starts in the cortex, and as it progresses abscesses almost invariably develop. In rare cases the change does not go beyond caseation. Tubercular deposits in the ureter are often met with; they

lead to considerable narrowing of the tube, and thus interfere with the passage of the urine and pus.

**Renal tumours.**—The chief are congenital cystic adenoma, papilloma, sarcoma, and carcinoma.

**Congenital cystic adenoma** is frequently very large, the outline of the kidney is never completely lost, though its natural uniform surface is often broken by projections. There is no distinction between cortex and medulla, and often the entire kidney substance is replaced by the new growth. In some cases areas of the cortex are intercalated between the cysts; the latter have smooth walls which are lined in the early stage with tessellated epithelium; they vary in size from a hemp seed to a grape. The trunk vessels are small. The ureter is attenuated, but pervious. According to Shattock, the cysts are developed from the meso-nephron (Wolffian body). The entire mass represents both meso-nephron and meta-nephron, or true kidney. Both kidneys are usually affected. The disease is sometimes associated with deformities such as spina bifida, talipes, and anencephalos. A similar condition of a portion of one kidney has been observed later in life, but the glandular element was more manifest.

**Papilloma** of the kidney is very rare. The growths, which spring from the wall of the pelvis, are in structure the same as villous tumour of the bladder.

**Sarcoma** is generally of the round-celled variety. It may occur at any age, but is more common in infants. In some cases it begins in the kidney proper, in others in the meso-nephron. In one case the kidney was found intact; the tumour entirely filled the abdomen. In structure it consisted of mixed round and spindle cells enclosing islets of corpuscles resembling glandular tissue. In another specimen there was perfectly striped muscular fibre.

**Carcinoma** occurs as a primary encephaloid growth in one kidney, or as a secondary deposit usually in both.

## CHAPTER LXXIX.

## ULCERS OF THE ANUS AND RECTUM.

**Ulcers about the anus.**—The principal varieties are : (1) Simple fissure ; (2) ulcerated syphilitic mucous tubercles (*vide* pages 42, 177) ; (3) epithelioma ; (4) ulcerated piles ; (5) ulcers in connection with fistulous openings and operation wounds.

Primary venereal sores are occasionally seen in this situation.

*Painful fissure of the anus* may exist alone, but it frequently starts from the base of a pile. It probably commences in an abrasion of the mucous membrane. The nerve fibres are subject to more or less constant irritation from contraction of the sphincter, and the tension upon them is greatly increased by the act of defæcation. It seems likely that the terminal twigs of the nerves are exposed in the floor of the ulcer, or it may be that they are constricted by the contraction of inflammatory lymph. An incision made in the axis of the fissure divides some of the muscular fibres of the sphincter, and with them the nerves ; a certain amount of physiological rest is given to the parts, and this promotes healing.

*Epitheliomatous ulceration* commences at the junction of the skin with the mucous membrane. The base of the ulcer is granular or tuberculated, and greatly indurated. On rectal examination it will usually be found that the structures beyond the anus are invaded, and the extent of this may be sufficient in itself to differentiate the disease from ulcerated piles.

*External piles* are rarely ulcerated beyond the surface ; and when internal piles are extensively

destroyed it is by a combined process of ulceration and gangrene, their bases being strangulated by the sphincter ani. Such a protruded mass shows an absence of marked induration; and there is no infiltration around the base, which is limited by the folds of the thickened anal mucous membrane and skin.

*Anal fistule* sometimes refuse to heal after being laid open. Then the margins and base of the ulcer become indurated, and the papillæ of the surrounding skin hypertrophied. This gives a warty appearance, something like epithelioma. But in epithelioma the induration is greater, and there is more outgrowth, the constructive process outstripping the destructive. The direction of an ulcer left after operation for fistula is suggestive of its origin. It must be remembered that long-continued irritation of a simple ulcer may cause it to take on a malignant character.

**Ulcers of the rectum.**—Those of greatest surgical importance are: (1) simple; (2) syphilitic; and (3) malignant. Tubercular ulcers are comparatively rare, often multiple, and are commonly accompanied by tuberculosis of other organs.

*Simple rectal ulcers* may be due to traumatic irritation; but their origin is generally involved in obscurity, since, when small and free from the disturbing influence of the sphincter, they give but little trouble. They are mostly situated within the lower two inches of the rectum. The floor or base shows but little induration; in fact, it may almost be as free from it as the healthy mucous membrane; but the margin is usually defined. An important sign, and one of especial value in the diagnosis of recent ulceration, is the bleeding occasioned by digital examination. This is usually slight, but one does not meet with it when the mucous membrane is intact. Rectal ulcers are slow to heal, on account of the muscular contractions of the bowel, and the irritation



caused by the passage of fæces. Whilst cicatrisation goes on at one part the ulcer often spreads at another. Other things being equal, the liability to stricture is proportionate to the duration of the ulceration. As the destructive process tends to spread in a circular direction, the resulting stricture is crescentic or annular.

*Syphilitic rectal ulcer* is in most cases a tertiary lesion ; but it sometimes appears during the secondary symptoms. Like other ulcers of the rectum, it is usually within reach of the finger. The occasional existence of scars about the anus has led some surgeons to conclude that the ulceration commences externally, and afterwards involves the rectum. This view at first sight appears strengthened by the fact that syphilitic disease of the rectum is many times more frequent in women than in men. (In women there is a greater liability of ulceration spreading from the genitals to the anus than in men.) The foregoing explanation may be true to a certain extent, but it does not apply to the majority of cases in which the ulceration undoubtedly begins internal to the sphincter. It is by no means rare for an inch or so of healthy mucous membrane to intervene between the anus and the seat of the disease. Reflex or sympathetic irritation of the rectum from disorders of the generative organs, and protracted constipation, cannot go for much in explaining the greater liability to syphilitic ulceration of the rectum in women ; for the same conditions would also predispose to malignant disease, and this is more common in males.

There is no certain sign by which we can tell simple from syphilitic ulceration of the rectum, so we always appeal to the history of the case, and seek for concomitant evidence of syphilis to make the diagnosis complete. Speaking generally, it may be said that syphilitic ulceration is more extensive, more rapid in



its development, and more amenable to specific remedies, than simple inflammatory erosion.

At the same time it is very inveterate, and gives rise to great discomfort from the pain, profuse discharge, and the secondary troubles consequent on stricture.

*Malignant rectal ulcer.*—Every form of malignant disease that attacks the rectum sooner or later causes ulceration. This either begins at the anus, from the disintegration of epithelial cancer, or quite clear of the orifice in connection with columnar epithelioma, or less commonly sarcoma. The base of the ulcer is very indurated, and of unequal depth at different parts, depressions alternating with hard nodular elevations. The margin has an irregular outline. It is raised, and the tissues are infiltrated with growth for some distance beyond it. The discharge consists of pus, slimy mucus, blood, and the débris of the new formation. It is very foul from decomposition, and the factor is in proportion to the rate of destruction of tissue.

Malignant ulcer of the rectum has a proclivity for the male sex. It is met with chiefly beyond mid-adult life. Stricture is a certain result; and the formation of a fistula is by no means rare.

## CHAPTER LXXX.

## STRICTURE OF THE RECTUM.

It has been already noted that simple, syphilitic, and malignant ulceration entails constriction of the calibre of the bowel. In the two former the cicatrization is purely inflammatory in nature. In cancerous disease it is partly due to contraction of the fibrous stroma, the same as the retraction of the nipple from atrophying scirrhous of the mamma.

Obstruction from cancerous stricture is increased by projecting nodules of the growth.

Stricture of the rectum, independent of ulceration, is very rare, but it may arise from the shrinking of plastic lymph effused into the submucous tissue, or from traction on the gut by the organised products of pelvic cellulitis. It is difficult to conceive how long-continued irritation can lead to localised annular hypertrophy of the muscular coat of the rectum without causing at the same time ulceration of the mucous membrane, or inflammatory thickening of the cellular tissue beneath it.

Stricture may be simulated by an unusually-developed fold or valve of Houston.

The stricture is crescentic or annular, according as the disease which causes it involves a part or the whole of the circumference of the gut. So long as a segment of the circle of mucous membrane remains free, the contraction is not likely to give rise to dangerous obstruction, save in the case of malignant disease, where a mass of new growth may block the passage at the seat of stricture.

**Effects upon the bowel above and below the stricture.**—The calibre of the gut for a long

way on the proximal side (it may be through the whole or greater part of the intestine) is very much *dilated*, and the muscular coat is enormously *hypertrophied* to compensate for the increased resistance. But, in spite of the hypertrophy, the bowel is distended with flatus, and scybalous masses collect in the upper part of the rectum and in the colon. *Constipation* is the natural result, but this may be masked by constant or periodical *diarrhœa* ("the diarrhœa of constipation"); for, in addition to the discharge from the ulcerated surface, the mucous membrane is fretted by incarcerated fæces, and so kept in a state of congestion and catarrh. From time to time a quantity of fluid is poured out to relieve the over-distended vessels. The hardened fæces are softened and diminished in size, they being churned, as it were, in the exudation. Then there comes, perhaps, a period of quiescence, to be followed, in turn, by another attack of diarrhœa. The important point to note is that all this time constipation may exist.

*Perforation of the bowel* sometimes takes place at a distance from the stricture from the pressure of hardened fæces, but more commonly immediately above it.

The perforation is either purely ulcerative, or ulcerative and gangrenous. I once performed colotomy for obstruction from malignant stricture of the rectum. At the post-mortem examination it was found that a mass of hardened fæces had, by its pressure, led to death of a considerable portion of the wall of the cæcum, and consequent perforation and peritonitis. The patient bore a healthy scar of amputation of the breast practised for scirrhus four years previously.

The perforation may take place into the peritoneum, or into an adjoining viscus, or it may set up abscess, and this may remain localised, or travel

along the bowel and open externally. In the last-mentioned event the fistula serves as an escape-pipe for the matters pent up above the stricture.

If the stricture is a purely inflammatory one, the ulceration often heals below whilst it spreads above, for the distal portion of bowel is in a state of rest compared with the proximal. Malignant ulcer never completely cicatrises on either side of the stricture. The traction exerted by the stricture, and the pressure of the exudation about it, cause obstruction to the circulation through the hæmorrhoidal veins; hence piles are likely to form. These, when indurated, as they often are, may be mistaken for cancerous nodules, especially as they are associated with ulceration and stricture.

Stricture entails straining at defæcation, and the mechanical effects of this are patulous anus, and in the female procidentia uteri, cystocele, and rectocele.

## CHAPTER LXXXI.

## TUMOURS OF THE RECTUM.

**Malignant tumours** of the rectum comprise squamous epithelioma of the anus, columnar epithelioma of the rectum proper, and sarcoma.

**Epithelioma of the anus** commences at the junction of the skin with the mucous membrane, and spreads deeply and widely in the surrounding tissues. It is essentially a new formation on the type of squamous epithelium, and it has the characters common to epithelioma of the lip, tongue, and cutaneous surface, the most striking feature being the presence of globes or pearls made up of concentrically laminated cells. As the growth encroaches on the mucous membrane, where the epithelium is columnar, cells of transitional form may be observed.

The inguinal glands are infected from the outward part of the growth, and the pelvic and lumbar from the rectal.

**Columnar epitheliomata of the rectum** are often so perfectly glandular in appearance that it is difficult, from a microscopical examination alone, to distinguish them from adenomata. (*Vide* Figs. 75 and 93.) But should a doubt arise as to the malignancy of a given specimen, it can be dispelled by the knowledge that the mode of growth is infiltrating and deeply destructive. The same is true concerning simple villous tumour and villous cancer of the bladder (page 440). "The boundaries between simple papilloma and villous cancer may be just as difficult to define as those between adenoma and carcinoma" (Billroth). When the glandular arrangement is well defined these rectal cancers are appropriately designated "adenoid." It will generally be

found that the superficial portions of the tumour conform more to the likeness of the normal anatomy of the mucous membrane than do the deeper.

The naked-eye and microscopical appearances of cancerous disease of the rectum vary as the rate of cell multiplication and the relative extent of the fungating and infiltrating modes of growth. In some cases there is a marked tendency to invade the sub-mucous coat and to develop a quantity of fibrous

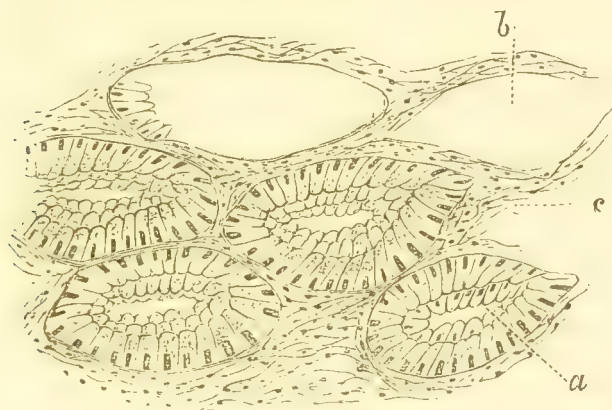


Fig. 75.—Columnar Epithelioma of Rectum.

*a*, Acinus lined by columnar epithelium; *b*, acinus from which the epithelium dropped out during the preparation of the section; *c*, interacinar stroma, formed of delicate fibres strewn with indifferent cells.  $\times 265$ .

tissue. The latter, as it undergoes cicatricial contraction, gives rise to great induration, and, as the hardening and proliferation are not uniformly diffused, the surface is tuberculated.

On the other hand, the interstitial tissue may be scanty, and richly strewn with indifferent cells, whilst the epithelial formation is abundant. At the same time the growth may spread more in the direction of the lumen of the bowel than in its walls, and consequently assume a tuberous form.

What was formerly described as "scirrhus cancer



of the rectum" is in reality nothing more than the indurated variety of columnar epithelioma. The term scirrhus as formerly employed to indicate simply a *hard* cancer is not inaccurate, but since it has been used in a more restricted sense—*i.e.* in reference to hard cancers starting in glandular and not in surface epithelium—its retention in the nomenclature of malignant diseases of the rectum is inadvisable.

**Sarcoma of the rectum** is less common than cancer. It either invades the bowel from without, or starts in the submucosa. Histologically, it consists of round and spindle cells and a variable amount of mucous tissue (myxo-sarcoma). Clinically and pathologically, it conforms to the laws regulating the growth of sarcomatous tumours in general.

**Innocent tumours of the rectum** are met with chiefly in the polypoid form, benign intramural growths being exceedingly rare. They are more common in children than in adults, contrasting in this way with malignant disease. The base of attachment is in most cases within reach of the finger. Every variety of simple polypus may be found. The following may be enumerated: (1) Fibro-glandular, or adenomatous; (2) villous; (3) vascular; (4) myxomatous; (5) warty. The foregoing classification is based partly upon the coarse anatomy and partly upon the microscopical structure of rectal polypi.

*Adenomatous polypus* occurs as a soft, fleshy mass, with a rather broad peduncle. The interacinose tissue consists of a more or less gelatinous ground substance, through which is woven a network of fibres, cell ramifications, and blood-vessels. In some cases it is exactly like the tissue that makes up the entire mass of a simple mucous polypus; in others it is more densely fibrous. It is richly vascular and cellular. But the characteristic feature is the presence of acini and tubular recesses lined with columnar

epithelium. The adenoid tracts are circular, oval, straight, or sinuous, and are distributed singly or in racemose clusters. The surface of the tumour is covered with the same kind of epithelium that lines the acini and crypts (Fig. 93).

*Villous polypus* is seen as an arborescent, leafy, or filiform outgrowth, sessile, or distinctly pedunculated. Histologically, it is composed of connective tissue in various stages of development—embryonic, mucous, and fibrous. The surface epithelial cells are columnar,

*Vascular polypus*, or *nævus*, is almost confined to early life; hence it is commonly known as the “vascular polypus of children.” It is found singly or in groups. It is often no larger than a pea or bean. It has a florid appearance, something like a raspberry. Structurally, it is made up of a delicate stroma of connective tissue, which supports the numerous large capillary blood-vessels. It is very similar to the small caruncular growths about the orifice of the female urethra. It is very liable to bleed.

Very rarely the veins of the rectum are enlarged so as to constitute a cavernous *nævus*. This condition must not be confounded with internal piles.

*Myxomatous or gelatinous polypus* has the same structure as common nasal polypus, with the exception that the epithelium is not ciliated.

*Warty polypus* is so seldom met with that a detailed description is not needed. The mucous membrane of the rectum over a considerable area has been found covered with small papillomatous proliferations.

The so-called “fibrous polypus” is generally a villous growth, or fibro-glandular tumour.

The chief symptoms of rectal polypi are tenesmus, sanguineous and mucous discharge, prolapse of the bowel, and reflex disturbance of the genito-urinary organs, such as irritation about the penis and frequent micturition.

## CHAPTER LXXXII.

## PROLAPSE, HÆMORRHOIDS, AND FISTULA.

*Prolapse of the rectum.*—In slight cases the loose submucous tissue just above the anus is stretched, and this allows a ring or fold of mucous membrane to protrude (prolapsus ani) ; but a more important variety is that where the entire thickness of the gut becomes prolapsed (prolapsus recti). This form is most common in children.

The causes are the same in each case—viz. laxness of the sphincter ani and muscular coat of the bowel, and mechanical strain upon the parts from long-continued or oft-repeated tenesmus. The irritation may arise from some rectal disorder, such as piles, polypi, or ascarides ; or, reflexly, from phimosis, stone in the bladder, enlarged prostate, or stricture. But, apart from reflex contraction, voluntary efforts to overcome obstruction to the flow of urine entail a bearing down upon the rectum.

In women the recto-vaginal septum may yield and form the wall of a broad-mouthed cul-de-sac (rectocele). The causes of this are loss of support from rupture of the perineum, and straining at stool. (*Vide* Stricture of the rectum, page 473.)

*Hæmorrhoids.*—The essential pathology of piles is a dilatation of the hæmorrhoidal vessels, and chiefly of the veins. When the vascular dilatation is limited to the skin around the anal orifice, the piles are called “external” ; and when it is confined to the mucous membrane of the rectum, they are termed “internal.” It very frequently happens that the two co-exist ; but even then the division is marked by a shallow groove on the surface, which serves as a guide to the surgeon

as to what should be tied and what excised in the operation of ligature for the cure of the disease.

In *external piles* the veins are dilated and their walls thickened, and the perivascular connective tissue is hypertrophied and indurated. The result of these structural alterations is the formation of firm nodular bluish-white masses, covered with thickened skin.

Loose folds of integument, radiating from the anus, are frequently seen apart from, or in continuation of, the hæmorrhoidal tumours.

In *internal piles* the same morbid changes are observed, but the arteries and capillaries often participate largely in the process.

On account of the arterio-capillary dilatation, the lack of surface support of the mucous membrane, and the liability to injury from the passage of hardened fæces, internal piles are prone to bleed.

In those cases where the ectasia mainly affects the veins, the piles are often pedunculated, and they present a somewhat livid appearance. Where, on the other hand, the arteries and capillaries are extensively enlarged, the piles are brighter in colour and more sessile.

Anything that serves to determine blood to the part, or mechanically prevents its return, conduces to the development of hæmorrhoids. Chemical or mechanical irritation of the rectum, and plethora of the portal circulation, are the principal points to be borne in mind. The ultimate causes are constipation, straining on account of difficult micturition, and alcoholism and high-feeding. Cirrhosis of the liver is an intermediate factor. Internal and external piles may become inflamed, and internal piles strangulated by the sphincter ani. When inflamed they are very tense, œdematous, and painful. The veins are thrombosed, and their walls and the connective tissue around are infiltrated with lymph and leucocytes.

Speaking generally, we may say that the morbid anatomy of piles consists mainly of venous dilatation and varicosity ; and that phlebitis is the essence of their inflammation.

*Fistula in ano.*—A complete fistula has one orifice on the cutaneous surface, and the other in the bowel. Of incomplete or blind fistula there are two varieties, internal and external. The former communicates only with the rectum ; the latter opens by the side of the anus. An incomplete fistula of either kind may become complete, and *vice versa*.

As a rule the internal opening is just above the sphincter, and the external near its outer border ; and the passage between them pretty direct. But there are many exceptions—*e.g.* the sinus, after “coasting the bowel,” may end on the opposite side (“horse-shoe fistula”) ; or from inefficiency of drainage the original tract may give off diverticula, or be extended for a long distance above the upper opening, or outwards from the external. In stricture of the rectum (page 472) the fistula joins the proximal dilated portion of the bowel.

Suppuration is the means by which the fistula is established. It may commence in an extra-rectal abscess, or in an ulcer of the mucous membrane.

The cause of such ulcer or abscess is either constitutional or local. The frequent association of fistula and phthisis suggests an origin in tubercular inflammation. Irritation from hardened fæces, or a foreign body, although it cannot often be proved to be an exciting cause, may still be such. I once removed a fishbone, and in another case a piece of a rabbit bone, each of which had transfixed the bowel immediately above the internal sphincter ; one end could be felt per rectum, and the other by means of a probe passed through the outer orifice of the fistula.

With regard to the propriety of an operation, it may be said that it turns upon the chances of the wound healing, and not upon the influence that a fistula has been supposed to have in checking the progress of tubercular disease of the lungs.

Experience teaches that the getting rid of a suppurating cavity or tract has a decidedly beneficial effect upon a patient suffering from phthisis.



## CHAPTER LXXXIII.

## PERITONITIS—APPENDICITIS.

PERITONITIS is most common as the result of injury or some other source of local irritation, such as substances derived from rupture of the underlying viscera or pathological cavities, or diseased states spreading by continuity—*e.g.* cancer of the liver, and typhoid and tubercular inflammation of the intestines. Peritonitis is usually described as acute or chronic, but between the two forms there are many intermediate degrees of intensity.

**Chronic peritonitis** is either general or partial. When general, it is caused by Bright's disease, or disseminated cancer, or tubercle. Partial peritonitis occurs in connection with localised disease of the structures beneath the serous membrane—*e.g.* an ulcer of the stomach or intestine, or inflammation of the pelvic organs; or, as the consequence of long-continued friction, notably in old hernial sacs. The lymph poured out from the vessels organises to connective tissue, which binds together adjacent parts, and, contracting, causes constriction of the hollow viscera, and various displacements. By continued stretching the adhesions may be elongated into fibrous bands, beneath which a portion of the bowel may slip and become strangulated. The peritoneum is greatly thickened, as is seen in hernial sacs, where the contents have been allowed to remain habitually prolapsed.

**Acute peritonitis** is set up by rupture, or ulcerative perforation of some viscus—*e.g.* the bladder or intestine; or by bursting of a hydatid cyst, or localised abscess; or by a wound from without, as in

the operations of gastrostomy, laparotomy, herniotomy, and cholecystotomy; or by infective inflammation spreading from the uterus along the Fallopian tubes.

The pathological appearances vary greatly in different cases according to the rapidity of the process, the general health of the patient, and as to whether the peritoneum be healthy, or altered in structure by previous disease. One result of chronic thickening of the membrane is to diminish the liability to acute general inflammation, so that the sac of an old hernia may be opened with less risk than that of a recent one.

The inflammation is termed serous, plastic, purulent, or hæmorrhagic, according to the prevailing product of exudation, the same as happens in pleurisy and pericarditis.

In surgical practice it is usual to speak of two varieties—sthenic and asthenic, or latent.

In acute sthenic peritonitis the signs and symptoms are most pronounced. They are severe pain; great abdominal tenderness; marked tympanites; knees drawn up to relax the tense abdominal muscles; fast, wiry pulse; and high fever.

In the latent or asthenic form there are the same physical conditions that underlie the above-mentioned symptoms, but the depressed or exhausted state of the patient prevents a general response to the absorption of the products of inflammation and the irritation of a wide tract of visceral nerves. One is often struck by the contrast offered by the comparative immunity from suffering and the revelations of the post-mortem room. An old man who has been operated upon for strangulated hernia may never rally sufficiently to manifest by general signs the profound structural alterations going on in the peritoneum.

**Morbid anatomy.**—Unless the peritonitis be caused by some irritant fluid poured more or less over the whole surface, the inflammatory changes are most marked at the seat of the original injury; *e.g.* whilst there is a copious deposit of lymph, and it may be pus, upon and about the contents of a hernial sac after reduction, the parts more remote may be only lightly glued together by a scanty fibrinous exudation. In some cases, however, and especially where a large wound has been inflicted, and the inflammation is of a septic nature, the latter spreads with great rapidity and soon becomes widely diffused—*e.g.* after ovariectomy.

The first stage is that of vascular injection; the subserous vessels are dilated, then there is exudation upon the surface of the membrane and into its interstices. As the natural secretion is highly fibrinogenous it is not a matter of surprise that, under the influence of the globulin-laden constituents of the blood, fibrin, which does not exude as such from the vessels, should be formed and deposited with great readiness. This really occurs before there is any striking alteration in the appearance of the membrane, and certainly before it has lost its lustre. But even at this early period it can be shown by gently drawing the coils of intestine apart, when it will be seen that instead of gliding over one another with the usual facility, they stick somewhat. Scraping, too, reveals the presence of a small quantity of semi-transparent glutinous matter. As the exudation becomes more profuse it collects in the furrows formed by adjacent coils of bowel. Here also the hyperæmia is more marked than elsewhere. In fact, on separating the coils, it will be found that, whilst their mutual pressure had prevented the accumulation of blood in the vessels at the surfaces of contact, it had as a natural consequence increased the tension in the collateral branches, and given

rise to the so-called *suction-bands* of congestion and exudation that skirt the confines of the more anæmic parts.

By this time the surface has lost its brightness, owing to the loss of the endothelial layer and the formation of fibrin. It looks minutely granular to the naked eye, and, with a low magnifying-glass, finely reticulate from the interlacing of fibrin filaments.

Authors differ as to the share that the serous endothelium takes in the process. Those who believe in the absence of formative activity from connective-tissue corpuscles, generally assert that here they become loosened by the exudation and softened by liquefactive degeneration; and that the richly cellular neoplasia consists mainly of fibrin and migratory leucocytes. They look upon the multi-nucleated giant-cells found in the exudation as masses of homogeneous plasma, embedding white blood-corpuscles.

Others state that the endothelial cells swell up and, together with their nuclei, divide and subdivide, and thus add greatly to the vascular effusion.

Meanwhile the subserous connective tissue becomes swollen, succulent, and infiltrated with liquor sanguinis and leucocytes. Nor does the process end here, for the muscular coat of the bowel loses its contractility, hence the tympanites from the unrestrained pressure of the gases within. It also softens, as shown by the readiness with which it tears on attempting to dissociate adherent coils of bowel. In pelvic peritonitis the bladder suffers in a similar way, and is unable to empty itself properly.

So far, only the plastic stage of peritonitis has been considered, but unless the inflammation resolves, or death of the patient supervenes at this period, further changes are observed. The exudation becomes more copious, and can no longer be retained upon the surface and within the meshes of the membrane.

The coating of lymph, sometimes distinctly laminated, is raised from its bed by the pressure of the fluid beneath. It can be readily peeled off, leaving a granular surface composed of loops of capillary blood-vessels surrounded by leucocytes. The outward pressure of the osmotic current separates the visceral and parietal layers and the involutions of the former. The fluid accumulates in the potential cavity, where it takes the course directed by gravity and least resistance; so it is found in greatest abundance in the pelvis, flanks, between the liver and the diaphragm, and beneath the liver. At first it is serous, or sero-purulent and cloudy, and generally contains some flakes of lymph washed off from the surface of the peritoneum. Then it becomes more turbid as the number of leucocytes increases, and the fibrin in suspension augments in quantity, and as both pass through retrograde granulo-fatty changes. Finally, it is quite purulent.

Some cases scarcely go beyond a plastic exudation, some stop with extensive serous effusion, whilst others pass rapidly into suppuration. The last is common in the asthenic peritonitis of old and exhausted patients, where the inflammatory effusion which often smears over the viscera, and resembles "melted butter," is more copious than one would suppose from the extent of the congestion observed post mortem.

Acute purulent peritonitis follows the admission of highly irritating matter, such as would be derived from extravasation of the gastro-intestinal contents, rupture of an abscess containing very infective (locally) pus, or the admission of air laden with septic germs. Under the last-mentioned conditions the softening of the peritoneum and subserous tissue is so great, and the vascular tension so high, that capillary ruptures are exceedingly common on the surface of the peritoneum, and into the lymph which

covers it and infiltrates its substance. This is known as *hæmorrhagic peritonitis*. The red corpuscles break up very rapidly, and the liberated hæmoglobin decomposes into other hæmatin compounds, so that the inflammatory products are variously stained. Where the extravasation is recent they are deep red and sanguineous; where it is older they are reddish-brown and ochre-coloured.

The fluid contained in the peritoneal cavity, which is highly decomposable, sometimes gives off gases of putrefaction. Inflation also happens when there is free communication with the stomach or intestines. In either case the tympanitic note is more uniform than when the hyper-resonance depends upon distension from gases imprisoned in the natural passages.

When gas is diffused in the peritoneal cavity, the anterior liver dulness is lost; this is owing to (1) accumulation of gas between the diaphragm and liver, and (2) to the falling back of the liver by its own weight through the aerial space. In a case of abdominal injury, disappearance of the anterior liver dulness is strongly suggestive of perforation of the stomach or intestine. An extremely distended colon may overlap the liver, and so increase the area of resonance, but rarely, if ever, to the extent of abolishing the liver dulness. In these cases, however, the outline of the bowel can often be more or less determined by inspection, palpation, and percussion.

**Microscopy of the exudation.**—The products of peritonitis will be found to vary as the intensity and duration of the inflammation. In simple plastic peritonitis there will be filaments of homogeneous fibrin, leucocytes, and swollen endothelial cells. In purulent hæmorrhagic peritonitis the fluid obtained from the cavity will contain flakes of fibrin in a state of granular degeneration, pus cells, granulo-fatty débris, blood corpuscles, pigment granules, and



sometimes crystals of hæmatoidin, cholesterine, and the fatty acids. Bodies of extra-peritoneal origin may also be discovered, such as the contents of hydatid cysts, of the gall bladder, and of the intestines.

**Vascularisation and organisation; adhesive peritonitis.**—As in inflammation of other tissues, new blood-vessels are formed. These are probably derived for the most part from loops and buds of pre-existing vessels; but it is quite possible that some have a separate origin in vaso-formative cells, and that they afterwards join the general circulation.

As the fluid is absorbed, the opposite surfaces adhere, and the organisation into connective tissue is the same in every respect as before described under *Healing of wounds* (page 213).

Although the peritoneum is very susceptible to the causes of inflammation, simple incised wounds heal very readily, provided the tension be not too great and the injured surfaces be kept free from all sources of irritation.

As plastic lymph is quickly effused, it is the object of the surgeon to bring the cut edges of peritoneum closely together, so that any communication with the external air or subjacent cavities may be shut off as early as possible.

The young connective tissue undergoes cicatricial contraction, and in this way many of the vessels are obliterated. Band-like adhesions become further attenuated, and even broken through, by the continued traction exerted upon them consequent on the movement of the parts. Thus, the pedicle of an ovarian tumour fixed in an operation wound may in course of time atrophy and lose its connection with the abdominal wall.

**Appendicitis** is the commonest cause of peritonitis in children and young adults. It is generally due to the irritation or obstruction effected by an

intestinal concretion, or a foreign body, such as a grape seed. The cul-de-sac may be distended and resemble a cyst ; its walls may be greatly thickened. When ulceration occurs, it usually ends in perforation, and an abscess is almost invariably formed. Peritonitis may be set up—(1) as the direct result of perforation ; (2) by extension of the inflammation around the appendix ; and (3) by the bursting of an abscess into the serous cavity.

## CHAPTER LXXXIV.

## STRANGULATED HERNIA.

THE symptoms of strangulated hernia are dependent more upon the nipping of the nerves and acute congestion of the vessels than upon obstruction of the bowel; for in some cases of obturator and femoral hernia, where only a part of the calibre of the gut is involved, and where there is consequently but partial obstruction, the local pain and the vomiting may be just as pronounced as when several coils are tightly constricted. The irritation of the visceral nerves is reflected through the abdominal "sympathetic" system, and shows itself as widely-distributed pain, reversed peristalsis, and increased secretion of the mucous glands.

Brinton explained the stercoraceous vomiting by direct onward contraction of the alimentary canal, and not by its reversal. According to this view, a circumferential current is established, which, on meeting with obstruction at the seat of strangulation, is turned back in the long axis of the bowel. This would be possible so long as the propelling tube remained full, but could not take place when it became practically collapsed and nearly empty. Moreover, experiments on the lower animals show that acute constriction is followed by reversed peristalsis; and no doubt this is what obtains in strangulated hernia.

**Mechanism of strangulation.**—When the neck of the sac is narrow and inextensible, and the surrounding structures are rigid, strangulation is effected immediately after the descent of the bowel; hence this is likely to happen in recent herniæ, and in old ones that have been kept habitually reduced by a

truss. On the other hand, where the neck of the sac is wide, unless several coils of intestine, or a single knuckle and a piece of omentum, are forcibly driven through the orifice, the symptoms are developed more slowly as the veins and capillaries become gorged with blood. As in constriction of a limb, the current through the veins is arrested before that in the arteries.

Subsequent serous effusion into the sac increases the tension.

**State of the bowel.**—In the first instance there is intense venous congestion, which causes the protruded gut to present a dark-red appearance. Then, if the tension be not relieved, it sets up inflammation. The effusion changes its character from serous to plastic, and the peritoneal coat loses its lustre from desquamation of the endothelial covering and coagulation of lymph on the surface. Meanwhile the walls of the gut are much thickened by congestion and exudation, and at the same time they are softened.

As the strangulation continues, stasis is followed by coagulation, and the parts, cut off completely from their vascular supply, lose their vitality and become gangrenous. When this stage is reached the bowel is black. Later, the blood-corpuscles are broken up and the colouring matter is discharged, and then there are dirty slate-grey patches. The condition is, in fact, typical of moist gangrene, and the tissues are so rotten that very little violence is necessary to break them down; hence the danger of forcible attempts at taxis. Rupture of the gut may take place into the sac or the abdominal cavity. In the latter case it gives way on the proximal side of the constricted coil; for, in addition to the uniform compression at the neck, there is the mechanical disturbance from peristalsis, whereas the bowel on the

distal side of the strangulation, after emptying itself of its contents, remains collapsed and comparatively quiescent.

The effect of distension and softening of the walls of the capillaries is occasionally seen in the form of *hemorrhage into the bowel* after reduction of the hernia. Such an event at first suggests ulceration into a large vessel, for the bleeding may be very copious (I have known half a pint of blood passed per rectum); but this is not the case. The capillaries of the mucous membrane, having lost their natural power of resistance, burst under the force of the arterial current let in upon them after the strangulation has been relieved. Extravasation to any great extent is very rare, whereas mucous and submucous ecchymoses are common enough.

**Fluid contents of the sac.**—In all cases of strangulated hernia there is a certain amount of serous exudation from the congested vessels of the bowel, or omentum, or whatever else may compose the hernia. Where the sac is large, and the rupture small, the fluid accumulation may be very extensive. The nature of the exudation varies with the degree and duration of strangulation; thus it is clear and straw-coloured; or pink, or deep red, from admixture with blood and dissolved hæmoglobin; or turbid from pus and flakes of lymph, the result of peritonitis; and lastly, it is sometimes mixed with the contents of ruptured gangrenous bowel, or the gaseous products of decomposition.

Apart from strangulation, the sac of a hernia may be dropsical and distended with clear serous fluid, when it simulates funicular and congenital hydrocele. This condition is known as "*hydrocele of the hernial sac.*"

**Artificial anus** is an unnatural opening of the bowel on the surface of the body. It is made

intentionally in the operation of colotomy. On cutting into the gut, if it has not already ruptured, the contents escape, and a communication is established with the lumen of the intestine on each side of the seat of strangulation. Extravasation into the general peritoneal space is prevented by previous plastic adhesion between the visceral and parietal layers of the peritoneum. The pressure of the underlying abdominal viscera, together with the peristaltic action of the bowel on the proximal side of the artificial anus, forces down the partition formed by the adherent walls of the proximal and distal segments of the gut. The outward flow of intestinal contents from the proximal segment keeps its orifice dilated, whilst the pressure exerted by the escape of fæculent matter, coupled with the projection of the partition above referred to (Dupuytren's spur), causes the opening of the lower collapsed portion of bowel to remain practically closed. The treatment of artificial anus consists essentially in getting rid of the spur-like barrier.

When the whole or the greater part of the intestinal contents escapes through the opening, it is called *artificial anus*; when only a minor portion escapes, it is termed *fæcal fistula*.



## CHAPTER LXXXV.

## INTUSSUSCEPTION OF THE BOWEL.

WHEN one portion of intestine is invaginated in another the condition is known as intussusception. It is by no means rare to find one or more such involutions post mortem, in cases where no symptoms pointed to their existence during life. It is doubtful whether they are formed before or after death, for peristalsis can be excited in animals by direct or indirect stimulation for some time after the heart has ceased to beat. These so-called "post-mortem invaginations" can be reduced quite easily. For their development there must be an irregular contraction of different portions of the bowel. Thus, if one segment is in a state of peristalsis whilst that immediately above or below it remains relaxed, the former can readily glide into the latter. The sudden increase in the calibre of the intestine beyond the ileo-cæcal orifice is the chief reason why intussusception is more common in this situation than elsewhere.

Children are more liable to it than adults, (1) because in them the general reflex excitability is very marked, and the bowel is readily excited to irregular peristaltic action through irritation of its nerves by ascarides, or other offending matter; (2) on account of the disposition of the peritoneum. It should be remembered that the part first invaginated always remains the lowest; or, in other words, that the ensheathed portion merely travels on, whilst the ensheathing tube is being continually rolled in; so that if the invagination commences at the ileo-cæcal valve, this will be found the most advanced part. Now, in children the cæcum is freely swung by the mesentery, instead of being fixed in the iliac fossa, as

it is in the adult, and consequently there is less check upon its involution.

A polypus, by dragging on the bowel, may start an intussusception.

The ensheathed portion of bowel acts as a foreign body, and stimulates the surrounding muscular walls to continued contraction. This may not cease until the ileo-cæcal valve has reached the rectum, or even protruded from the anus. The attachment of the mesentery hinders the descent of the bowel chiefly at its posterior aspect, so that the orifice of the invaginated tube looks somewhat backwards. This should be borne in mind on rectal examination, as otherwise the prolapse might be mistaken for a polypus, from which, however, it differs in not being fixed at one spot.

The symptoms are dependent on the degree of strangulation, and not on the simple existence of invagination, just as in an ordinary external hernia.

#### **Changes secondary to strangulation.—**

The blood-vessels of the prolapsed gut become engorged, then inflammation sets in, and the part is much swollen. The mucous membrane secretes a quantity of thick mucus, which, together with some blood derived from rupture of capillaries, is often discharged from the anus. Lymph is thrown out on the opposed peritoneal surfaces, which it glues together after the process of invagination is complete. As the result of acute strangulation the ensheathed bowel may slough away. Extravasation of intestinal contents into the peritoneal cavity is prevented by plastic exudation uniting the contained and containing portions of bowel at the base of the invagination. But before the gangrenous part has time to separate, the patient usually dies from collapse or general peritonitis; and where it is effected there is the certainty of an annular stricture from cicatrisation of the inflammatory products.

## CHAPTER LXXXVI.

## TUMOURS.

“A TUMOUR is a mass of new formation that tends to grow and persist.” This *tendency* is in marked contrast to that of inflammatory neoplasia, which is to arrive at a typical termination. Fatty tumours, it is true, frequently become arrested in their growth, and papillomata, as in the case of common warts, often disappear spontaneously ; but still the tendency is the other way. Again, although long-continued irritation may certainly be the exciting cause of a tumour, as in the case of epithelioma of the lip and tongue from friction against a pipe or tooth, and in the same disease sometimes starting at the seat of old chronic inflammatory lesions, such as syphilitic ulceration of the tongue and skin, yet, whilst we can produce inflammation at will, we have no absolute power of producing a tumour at all.

In studying the anatomical basis of tumours, it will be found that they all have their “type in some natural tissue of the body, either in the embryonic or developed state.” In other words, a tumour is not a parasite ; there is nothing truly alien in its nature ; it is the rebellious scion of a parent stock. If the elements of a new growth are like those of the tissue where they are found, they are said to be *homoplastic*—*e.g.* osteoma growing from bone ; if they differ, they are termed *heteroplastic*—*e.g.* enchondroma of the testicle. From a knowledge of the fact that any anatomical or physiological peculiarity of a tissue is usually stamped upon the tumour springing from it, a great insight as to the probable nature of the latter may be obtained ; thus one would expect osteoma,

enchondroma, and sarcoma to be frequently connected with bone, myo-fibroma with the uterus, and so on. It is also clear why myeloid sarcoma should have its seat almost exclusively in bone, and why sarcoma of the choroid should be melanotic, and glio-sarcoma of the retina and nerve centres present a stroma, like that of the natural structure of these parts.

A tumour is malignant in proportion to the rate at which it destroys the tissue and tends to shorten life.

It is impossible to denote definitely where malignancy ends and benignancy begins, for not only are there forms of intermediate gravity, but in the history of a given growth it may so alter in character as scarcely to be described by the same name—*e.g.* a “recurrent fibroid” may at first consist largely of fibrous tissue, but each successive recurrence after operation is often marked by a great increase in the number of cells.

The **signs of malignancy** are :

(1) Local recurrence after apparently complete removal.

(2) Dissemination or generalisation in other parts, either directly by associated lymphatics, or more remotely by the blood-vessels. Multiplicity of growth may in some cases be explained as a general outbreak from a common source rather than a causative sequence. It is interesting to note that the secondary growths maintain the type of the primary. Thus, alveolar cancer of the breast is reproduced in the lymphatic glands, liver, and other parts; columnar epithelioma of the rectum, in the liver; squamous epithelioma of the lip, in the cervical lymphatics; melanotic sarcoma of the skin and colloid cancer of the omentum or ovary follow the same rule.

(3) Infiltration of surrounding tissues by the elements of the tumour.

(4) Rapidity of increase, leading to destruction by

ulceration or sloughing. The vital activity of the cells, being expended more in multiplication than in development, leads to a deficient vascular supply, and great liability to suffer from injury.

(5) Multiplicity, though suggestive of malignancy, does not necessarily imply it, for some benign new formations (*e.g.* fatty tumours, warts, fibrous polypi of the skin, and atheromatous cysts) are often multiple.

(6) An apyrexial cachexia from impaired assimilation leading to general wasting.

(7) The influence of heredity is certainly more marked in malignant than in simple tumours.

(8) Direction of growth. Benign tumours springing from a surface are centrifugal; malignant, both centrifugal and centripetal. Compare the mere outgrowth of a papilloma with the deeply-rooted invasion of an epithelioma.

**Classification of tumours.**—Formerly, when tumours were arranged on a clinical basis, such terms as “medulloma,” “encephaloma,” “soft cancer,” etc., were applied without distinction to any soft, rapidly-growing new formation. In like manner, the word “melanoma” was used to indicate a tumour containing pigment. It is true the other attributes were implied, but it was none the less unscientific, for a soft wart on the skin may be as black as a virulent melanotic sarcoma. We now employ an anatomical classification, for it serves to point out the resemblances and differences of allied growths, and to distinguish between their essential nature and their subordinate nutritive modifications. Relying upon the microscope, there is no fear of mistaking a disintegrating infarct for a malignant tumour.

## CHAPTER LXXXVII.

## THE FIBROMATA.

WHITE fibrous tissue exists in abundance in many tumours. In some—*e.g.* the fibro-adenomata—the fibrous element far outstrips the glandular. The term fibroma denotes the absence from the growth of any more characteristic structural constituent.

The fibromata rank amongst the benign tumours. They arise from connective tissue, especially the subcutaneous and submucous, but they are met with in many other parts. The so-called “fibroid” tumours of the uterus belong more strictly to the myomata, with which they will be considered. We shall divide the fibromata into three groups: (1) The isolated encapsuled tumours situated beneath the surface; (2) the more widespread overgrowths of the cutaneous and subcutaneous connective tissue, known as hypertrophies or diffuse fibromata; (3) fibrous polypi. It may be stated generally that when fibrous tumours commence either in the skin or in the submucous tissue, the tendency is for them to become pedunculated.

The fibromata naturally fall under two heads, according to their consistence and vascularity. Thus, we speak of the firm and soft varieties.

In structure the fibromata present the same variations of textural arrangement as attains in normal white fibrous connective tissue. The greater portion of the tumour consists of bundles of fibres interwoven in various directions. The general disposition of these bundles is along the course of the blood-vessels; very often they form concentric layers around the latter, giving the section a lobulated appearance, and this is more marked when the bundles between the lobules are less closely packed than those which



constitute the latter. In some cases the fibres are loosely interlaced, forming an open meshwork ; in others they are so intimately connected as partially to obscure the fibrillation. The fibrillation itself varies much, so that microscopical sections appear like spun glass, or present wavy fibres with definite outline. Elastic tissue is usually absent. The cells show great diversity in number, size, and shape in different tumours, and in different parts of the same tumour. The younger the tissue, too, the larger and more numerous the cells ; in fact, it may be difficult in the earliest stages to distinguish between a developing fibroma, a myoma, and a spindle-celled sarcoma. For the most part, however, the cells are small. They are round, angular, fusiform, or branched. Some elongate, and become finally lost amongst the fibres with which they blend. We have examined specimens where not a single cell could be seen. On the whole, the fibromata are scantily supplied with blood-vessels ; some appear almost devoid of them. The softer forms are more vascular, and are sometimes permeated by a cavernous network. When wounded or ulcerated, the fibromata bleed freely, since the vessels are fixed in dense unyielding tissue—canalisation.

**Secondary changes.**—The most constant is calcification, either confined to the septa or as a petrification of the entire tumour. Next to this comes mucoid softening. True cystic formation is rare. The skin may ulcerate over the fibromata, and the inflammation spread into the interior, but they are not subject to primary suppuration.

The **firm fibromata** are very dense, creaking on being cut with a knife. The cut surface looks dead-white and coarsely fasciculated, or greyish and glistening, according to the degree of homogeneity of structure.

They grow from fasciæ, bone, periosteum, nerves, and other parts. Well-marked examples are the *fibrous epulides* of the jaws, which spring from the bone or periodontal membrane, not from the mucous membrane of the gums. They project by the side of the teeth, which frequently become loose. When they ossify (and this is not rare), the bone is deposited in the direction of the vessels—*i.e.* from the point of attachment towards the periphery. Ossification may advance to within a short distance of the surface, which is covered by mucous membrane. If completely removed there is almost entire immunity from recurrence.

What are known clinically as *neuromata* consist either of mucous or of firm fibrous tissue. (*Vide* Myxomata, pages 523-6.) The fibrous neuromata, like the mucous, start from the connective tissue around the nerves and between the fasciculi. The nerve-fibres are stretched over the tumour. We have seen a fibrous neuroma the size of an egg. Under the microscope it showed minute cells distributed with great regularity through a delicately fibrillated stroma. Some nasal polypi belong to the firm fibromata.

The **soft fibromata** include the diffuse fibrous hypertrophies of the subcutaneous tissue, and many pedunculated tumours attached to the skin and mucous membranes. The *diffuse fibromata* consist of loosely woven bundles of connective tissue, in the interstices of which the vessels ramify; the latter are sometimes very large. They form doughy masses, which sometimes hang in overlapping folds from the buttocks, thighs, and other parts of the body. From its structure and consistence the new formation was called *molluscum fibrosum*, or *fibroma molluscum*, a name still in use, but now generally restricted to the fleshy fibrous polypi of the skin.

When the subcutaneous soft fibromata project

from the skin as sessile or broadly pedunculated masses they are known as *wens*. The term *wen* is also used to denote a sebaceous cyst. Like the other diffuse fibromata, they have no capsule.

*Elephantiasis Arabum* bears a marked structural resemblance to diffuse fibroma, but instead of being a simple non-inflammatory overgrowth, it appears to be closely connected with, if not dependent upon, recurrent attacks of lymphangitis. The scrotum and legs are the parts usually affected. The growth often assumes enormous dimensions, weighing as much as forty or fifty pounds. It is mostly found in Orientals, and in them "is now generally recognised to be ultimately due to the presence of *Filaria sanguinis hominis*, a parasite which infests, besides other places, the lymphatics of the scrotum and lower limbs, and gives rise to inflammation, and obstruction of the lymph-current" (Ziegler).

Other causes of irritation of the skin may lead to "acquired elephantiasis."

Circumscribed, encapsuled, soft fibromata are met with in the subcutaneous tissue, but they are much rarer than the diffuse variety.

**Fibrous polypi** occur on the cutaneous and mucous surfaces. They may be considered as localised overgrowths of the subcutaneous and submucous tissues. Those growing from the skin have been referred to under the name "*molluscum fibrosum*." They are usually multiple. Their number may in some cases be counted by tens and hundreds. In size they vary from a pea to a potato. T. Fox describes two forms, the simple and the fungoid; the latter are very vascular.

Soft fibrous polypi of the mucous membranes are met with in the uterus, vagina, stomach, and intestines; but those of greatest surgical interest grow from the *nasopharynx* and contiguous sinuses (frontal,

sphenoidal, etc.). Clinically, many of them are more nearly related to the sarcomata than to the fibromata, for they tend to continuous growth, absorb everything in their way, and often recur after removal. Others, however, are quite innocent in their nature, and between the two extremes there is every gradation.

**Keloid.**—Keloid is a rare form of fibroma of the skin. It is said to occur as an idiopathic affection, when it is termed *true* to distinguish it from the so-called *false* variety, which grows from a cicatrix. Doubts have been cast upon the theory of an intrinsic ætiology, some authorities contending that each form owes its origin to local irritation. Keloid consists essentially in hypertrophy of the corium. In the early stage of its growth there is cell infiltration along the vessels. Eventually coarse bundles of connective tissue are formed, which give rise to a tuberos, discoid, or band-like elevation of great density. The central mass not uncommonly sends out claw-shaped radiating processes. Additional deformity is caused by the traction exerted on the surrounding skin. In so-called true keloid—and in this way it differs from cicatricial keloid—the papillæ remain, but the epidermis is usually thinner than normal. It is said to contain neither hair follicles nor sebaceous glands.

The disease under consideration known as Alibert's keloid is quite distinct from Addison's keloid, which is morphœa.

## CHAPTER LXXXVIII.

## THE LIPOMATA.

THE majority of fatty tumours are met with between the ages of thirty and fifty, when there is a general disposition to obesity. The local determination of the growth can in many cases be referred to some injury or long-continued friction. They are occasionally hereditary. They are situated chiefly on the trunk, especially about the shoulders and waist; parts subject to the pressure of articles of dress, such as the braces, corset, etc.; here they spring from the subcutaneous fatty tissue. Collections of fat are seen now and again in the synovial fringes of joints (Fig. 49) and tendon sheaths, resembling the appendices epiploicæ. Fatty tumours mostly occur singly, but several may be found in the same subject, the more so when hereditary.

**General anatomy.**—As a rule they form roundish projections on the surface of the body. The most constant feature is lobulation; this may be visible, or be made out only on manipulation, or after dissection. When pressure is made on the skin at the margin of the tumour, that over the surface becomes dimpled, from stretching of the interlobular bundles of fibrous tissue that are fixed to the skin.

The author has seen a polypus as large as an orange attached by a narrow stalk to the buttock; one half of the structure was composed of fat cells intermingled with the other, which consisted of dense fibrous tissue.

The lipomata are usually circumscribed and limited by a distinct capsule, which is fixed to the surrounding tissue by a loose meshwork and strong bands of

fibrous tissue. The latter conduct the blood-vessels, which enter chiefly at the upper and deeper parts. The external attachment may be so slight that the tumour changes its position; the resistance met with in the subcutaneous cellular tissue being slight, the force of gravity is sufficient to cause the displacement.

Very rarely fatty tumours show no limitation, the fat composing them being continuous with the normal tissue around (diffuse lipoma). The most common situations are those beneath the chin and back of the neck.

Lipomata are occasionally found connected with the peritoneum by a prolongation of the latter through a cleft in the abdominal wall, particularly at the linea alba; they then grow from the retroperitoneal fat or from herniated portions of omentum. The possible danger attending their removal is obvious.

Fatty tumours vary in consistence. They may be so soft and elastic as to simulate fluid collections; chronic abscesses and sebaceous cysts have been mistaken for them. On the other hand, they may be very firm, resembling fibrous tumours. The lobules are separated by areolar tissue, in which the supplying vessels are embedded. The amount of this, together with the composition of the fat in a given case, explains the density. In colour they are bright yellow, or yellowish-white. Like the normal fat, their yellow colour is deeper in dark-complexioned people. In children especially they often contain a considerable quantity of erectile tissue, so that by pressure their size can be much diminished; and they bleed freely on removal (nævroid lipoma).

Compare a fatty tumour after enucleation with an enchondroma of a long bone or the pelvis, and an agglomeration of enlarged lymphatic glands in Hodgkin's disease. All these are lobulated, but the



enchondroma is very hard, of a bluish-white colour, and is attached to bone. The glands are not so yellow as a fatty tumour, but they are firmer, and large trunk vessels can usually be seen passing through the mass, for it is usually taken from the abdominal, mediastinal, or cervical regions.

Lipoma forms compound tumours with myxoma, sarcoma, angioma, and fibroma.

**Microscopy.**—The greater part consists of fat cells, which do not differ from the normal, except that they

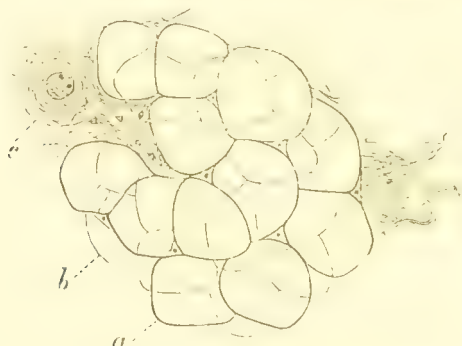


Fig. 76.—Fatty Tumour.

The cells, *a*, exactly in focus, have a more strongly marked outline than those, *b*, immediately beneath them; *c*, blood-vessel; connective-tissue corpuscles lie between the fat cells.  $\times 265$ . The section was cleared with oil of cloves.

are somewhat larger. They are roundish or polygonal from mutual pressure. The cells just out of focus are seen in dim outline (Fig. 76). Bundles of areolar tissue and blood-vessels run between the lobules. Fine fibres and connective-tissue cells may be found between the individual fat cells. This last phenomenon is explained by the *mode of growth*, for the cells do not contain fat at first; this is stored up later. The first cells of the tumour are derived from the connective tissue, and the tumour increases in size by the addition of new cells produced by vacuolation and division of pre-existing

ones, and possibly also by the fixation of wandering cells. The interlobular, and, to some extent, the intralobular, tissue is fibrillated.

**Secondary changes.**—Fatty tumours are not very prone to structural modifications.

Calcification and even ossification are occasionally met with in the matrix. The fat may alter its composition and become semi-fluid. The fatty acids may then be set free and crystallise; this is often produced by the action of reagents used in the preparation of microscopical sections. The skin over them may be atrophied by the continuous pressure, and this, together with accidental injury, may cause ulceration of the mass. Central suppuration rarely or never occurs.

Fatty tumours neither generalise nor recur locally after removal.

## CHAPTER LXXXIX.

## THE ENCHONDROMATA.

THESE tumours, which have their type in cartilage, grow for the most part from bone, and that, too, in the vicinity of diarthrodial joints and synchondroses.

They are not, however, confined to these situations, but are found in tissues (*e.g.* the parotid gland and testicle), in which at no period of their natural existence is any trace of cartilage to be seen. They are said not to spring from cartilage. It must be borne in mind that cartilage is a primary derivative of embryonic tissue, and hence it is not surprising that it should be developed where the latter forms the structural basis of new growths.

The mixture of cartilage with embryonic and mucous tissue is thus readily explained, and hence the varieties, chondro sarcoma, chondro-myxoma, etc.

Most enchondromata are innocent in their nature, rarely giving rise to secondary deposits. When these occur it is chiefly in the lungs. If all the indifferent cells are used up in the production of cartilage, the tumour may fairly be designated an enchondroma; but where there is only a limited conversion, it is better to speak of it as a chondrification, for the intrinsic nature of the growth is not modified thereby. A sarcoma is none the less malignant because islets of cartilage are scattered through its substance. We have seen this in a testicle removed during life (Fig. 65). The patient died within six months, with secondary deposits in the lumbar and mediastinal glands, and in nearly all the abdominal viscera. In another case the association of sarcoma with enchondroma was delayed; but what appeared at first to be an innocent tumour of the

parotid (for it only attained the size of a small orange in twelve years) afterwards made such progress that it doubled its size in six months; one-half was composed of hyaline chondro-myxoma (*vide* Fig. 77), the other of large round-celled sarcoma.

It may be said, then, that cartilaginous tumours of parenchymatous organs should always be regarded

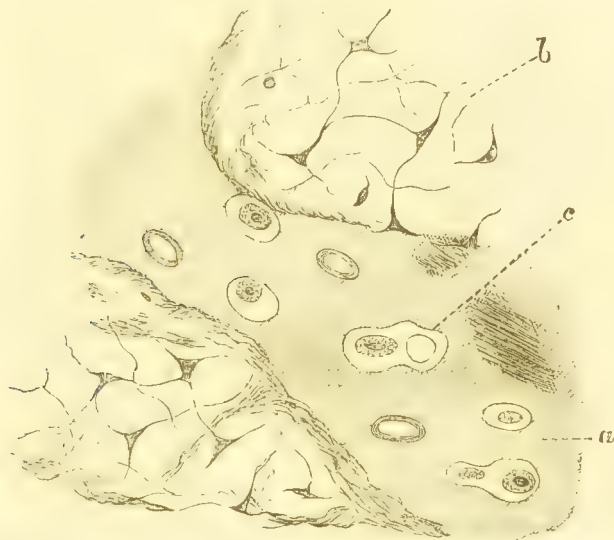


Fig. 77.—Myxo-Chondroma of Parotid Gland.

*a*, Hyaline cartilage; *b*, mucous tissue, with stellate corpuscles and slightly fibrillated gelatinous matrix; *c*, cartilage capsule, containing a nucleated cell and a fat globule.  $\times 265$ .

with suspicion. The same is true of those growing from the shafts of bones and the jaws.\*

**Classification.**—Enchondromata may be divided into three groups: (1) Those growing from the metacarpal bones and phalanges; (2) those springing from the ends of the long bones and the pelvis; (3) those found in soft tissues—*e.g.* the parotid gland, testicle, and lungs.

\* *Vide Lancet*, Nov. 24th, 1877.

1. *Metacarpal and phalangeal enchondromata* are often multiple. They commence in the interior of the bones, usually at the ends. They seldom grow larger than a walnut. They may calcify, but they do not ossify. Their surface is smooth, or only very slightly lobulated. The tumour is encapsuled, and consequently its outline is sharply mapped off from the encasing

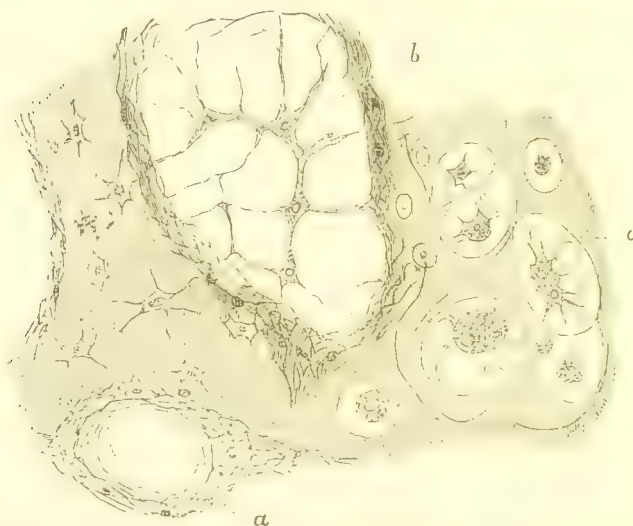


Fig. 78.—Cystic Ossifying Enchondroma of the Diaphysis of the Femur, from a Girl *æt.* 17.

*a*, Cyst bounded above by an osseous bar; *b*, mucoid softening of matrix, anastomotic stellate corpuscles form a meshwork through the space; *c*, hyaline cartilage, the capsules are distended with mucin.  $\times 265$ .

bone, which becomes expanded and absorbed by the pressure of the growth. They select by preference the periods of childhood and youth. They are perfectly benign. The matrix is hyaline, or faintly fibrillated, and the cells are comparatively small, and polymorphous.

2. *Enchondromata springing from the ends of the long bones and the pelvis* often attain an enormous size. Coarsely lobulated like fatty tumours, they

differ from them in every other respect, being hard and of a bluish-white opalescent appearance.

They grow from the surface of the bones, which they absorb by pressure and destroy by metamorphosis of tissue. They are encapsuled on the surface, but their structure is continuous with the osseous tissue. Coarse fibrous bands carrying the blood-vessels intervene between the lobules. Nutritive changes are very common, calcification and mucoid degeneration taking the lead. The former is recognised as dead white patches of irregular shape between the semi-translucent nodules of cartilage. The latter causes softening, and so large anfractuous cavities. They may ossify, or remain free from this and all other secondary modifications of structure.

In the pelvis they are attached near the symphysis or the sacro-iliac synchondrosis.

Unlike the smaller phalangeal variety, they tend to indefinite increase in size, but they resemble them in being generally benign.

3. *Enchondromata of the soft tissues* are seldom simple. Thus, mixed "parotid tumour" may contain cartilage along with glandular, mucous, or sarcomatous tissue, one or more of them (Fig. 77). In the testicle sarcoma is rarely absent.

The degree of malignancy is dependent upon the amount of the embryonic tissue.

**Histology.**—The matrix is hyaline or fibrous; or it looks like spun glass. Considerable variation in this respect may be met with in the same tumour. If calcified, microscopical sections look granular and opaque by transmitted light, but the capsules and cells can again be brought into view by the solvent action of a dilute mineral acid. The cells are round, oval, angular, or multipolar, with long branching offshoots. The protoplasm may be clear, or obscured by fat granules and drops of mucin (Fig. 78).



The entire cell may be petrified along with the matrix.

If lobulated, the mass is intersected by vascular fibrous tracts, thus differing from normal cartilage. The blood-vessels furnish the lime salts for calcification, and hence the infiltration is more advanced at the periphery of the lobules and between them than in their interior. In mixed tumours—*e.g.* chondrosarcoma—the cartilage is sharply mapped off from the surrounding tissue (Fig. 65).

## CHAPTER XC.

## THE OSTEOMATA.

THE osteomata proper are bony growths, not dependent upon precedent inflammation.

Deposit of bone is an accidental or an integral part of many morbid processes: thus it is found as a nutritive modification in many tumours, especially those springing from the osseous framework—*e.g.* the sarcomata, enchondromata, and fibromata. Bone is usually present in subperiosteal sarcomata; it is far from rare in the large lobulated cartilage tumours attached to the ends of the long bones and the pelvis; and it is almost constant in fibrous epulides. Then, again, it is one of the final anatomical products of inflammation of bone. In cases where the erosion has been local, and the loss of substance made up by protuberant granulations, the latter may ossify *en masse*, and the result simulate the true non-inflammatory exostoses; but, on section, the base of the new formation is found not to be limited by the surface level of the bone, but to lie some distance in the interior.

As in the osteomata proper, the Haversian canals of these inflammatory osteophytes lie at right angles to those of the old bone; for ossification follows the direction of the capillary loops in the outwardly directed granulations.

*Muscles and tendons* subjected to much strain or irritation are now and then extensively ossified, the bony deposit commencing as a rule next the points of attachment; *e.g.* in the deltoid and biceps muscles of infantry soldiers from pressure of the butt end of the rifle, and in the adductors of the thigh of cavalry,

the "rider's bone" (Rokitansky). But in these cases there must be an inherent disposition to bone formation, else it would be more common than it is.

In like manner the bones are sometimes the seat of multiple outgrowths at the points of origin and insertion of the muscles, without there being any ascertainable reason (O. Weber). Probably these cases are analogous to those of *myositis ossificans*, a disease which usually begins in childhood. It is a slowly progressive inflammation which attacks the muscles of the trunk and limbs, but spares those of the diaphragm, heart, larynx, and œsophagus. The muscular elements waste, and their place is taken by plates of newly-formed bone. It is excessively rare. The affection is quite distinct from multiple hereditary exostosis, in which the bony outgrowths are chiefly situated about the epiphyses, are rounded, and covered with cartilage.

In *old age* there is a marked tendency to ossification in certain tissues; *e.g.* the cartilages of the larynx, trachea and bronchi, the costal cartilages, and intervertebral ligaments. This is interesting, as showing that, whether the bone formation occurs as a new growth or a nutritive modification in pre-formed tissue, it is evidence of a physiological degeneration, and serves to explain the apparent anomaly of bony retrograde metamorphosis occurring mostly in old age, whilst the osseous tumours are rarely met with beyond mid-adult life. Although bone is formed in these cases, the change is largely one of mere calcification.

The plates of bone found in the *meninges* of the brain and cord are probably the consequence of chronic inflammation.

Cornil and Ranvier say they have seen true bone in the calcified adventitious cysts of *hydatid tumours*, but this must be very rare. The author has observed

a nodule of bone in the centre of a simple *bronchocele*. Bone is one of the numerous contents of *dermoid cysts*.

The osteophytes thrown out around the joints affected with *tumor albus* and *dry rheumatic arthritis* are clearly irritative and inflammatory. In *ataxic arthropathy* they are now and again met with as part of a trophic articular lesion ; but M. Charcot informed me that they are quite the exception in this disease, and that he was uncertain whether they grew from cartilage or not.

Having cleared the way of these irregular and secondary bone formations, the course is open for the consideration of :

The **osteomata**, which almost invariably arise from pre-existing bone. The vast majority occur as outgrowths, or *exostoses*. A few occupy the interior—*e.g.* the medullary cavities of the long bones, *enostoses* ; but even these cause projections from the surface in most instances, and are sometimes of inflammatory origin. Like normal bone, the osteomata vary in density, and it is customary to divide them into two groups, the *compact* and *spongy*. The former includes the ivory osteomata, and those that resemble in consistence the outer part of the shafts of the long bones.

The **ivory osteomata** are mostly situated on the flat bones of the skull, and usually on the inner surface. They are found as plates, or roundish nodules, composed of concentric laminae parallel to the surface of the mass. They are found not to contain blood-vessels. If the latter existed at an earlier period of development, they must have become obliterated by the pressure of the bony deposit around them, in much the same way as syphilitic cranial osteophytes are rendered extravascular.

The bone corpuscles in these tumours have long outrunners, which are for the most part directed

towards the surface, like those of the crista petrosa of the tooth fangs (Cornil and Ranvier).

Compact osteomata are found in other situations than the cranium. In the College of Surgeons' museum is a fine specimen attached to the lower jaw.

The **spongy osteomata** range in density from an open cancellous fretwork to a closely set trabecular structure bordering on the compact variety. They grow chiefly from the ends of the long bones in the vicinity of the epiphyseal cartilages. In all probability they start from the latter, or at least from the superjacent periosteum. For this reason their origin is limited to the term of existence of epiphyseal growth of bone. The spongy osteomata are occasionally met with at the rough muscular attachments on the diaphyses; *e.g.* in the femur near the opening in the adductor magnus, and in the humerus at the supracondylar ridges. (In some animals normal bony projections exist in these situations for the support and protection of the large vessels and nerves.)

The epiphyseal osteomata are in intimate connection with the cancellous tissue of the ends of the bones. They consist of rounded or lobulated masses. They have a surface layer of hyaline cartilage, from which they continue to grow, and this is covered with periosteum. *Bursal cysts* not uncommonly develop over them, and by some pathologists it is asserted that these from the first are continuous with the articular synovial membranes, and thus constitute a source of danger attending operations upon the tumours in question. On account of their cartilaginous investment the spongy osteomata are sometimes spoken of as *ossifying enchondromata*; but this is not quite correct, for when they cease to grow the surface cartilage ossifies—a circumstance, as far as my observations go, that never obtains in undoubted enchondromata. During their development

the cartilage capsules are dissolved, and the proliferating cells contained within are set free to form a layer of embryonic tissue, and in this the ossification goes on. Whilst the deeper portion of the cartilage is encroached upon and used up, the superficial layer is constantly being added to.

**Subungual exostoses** are spongy osteomata found for the most part beneath the nail of the great toe. Owing to the pressure over them suppuration and ulceration often occur, and the subsequent projection of inflammatory granulations from beneath the nail has led to their being mistaken for sarcomata.

**Microscopy.**—Sections made vertical to the surface show from without in (1) a fibrous layer, richly corpuscular, next the cartilage; (2) a narrow band of hyaline cartilage; (3) a stratum of osteogenic granulation cells; (4) fully-formed bone. The last has all the characteristics of true bone—viz. Haversian canals, lacunæ, and canaliculi; but the concentric lamination is less perfect than in the normal tissue.

**Hereditary multiple osteomata.**—These form a notable exception to the rule that osseous tumours are rarely seen in children under ten years of age. Hereditary or not, the osteomata as a group are not uncommonly multiple; but the variety under notice affects many bones of the body, and that, too, at a very early age. Fig. 79 is a copy of a photograph of a boy aged ten years, in whom the disease is well marked. His father and brother were affected in the same way, though to a less degree. The tumours first appeared when he was very young. They were extremely hard, and greatly interfered with the movements at the joints. Specimens taken from a similar case, in which nearly all the bones in the body were involved, presented a nodulated surface. The majority were seated near to or on the epiphyses, although there were several exceptions to this. Some were





Fig. 79.—Multiple Hereditary Exostoses.

pedunculated, others had broad bases of attachment; all were covered with translucent cartilage. They would seem to be the outcome of an excessive and ill-directed osseous development, comparing in this way with congenital growths, *e.g.* *nævi*.

The osteomata are benign: they do not recur after complete removal; and although sometimes multiple, they do not generalise. After a time they cease to grow. In themselves they are painless. They cause annoyance by their unsightliness, by the occasional occurrence of inflammation and ulceration of the skin over them, and by their impeding movement.

The **odontomata** are made to include inflammatory exostoses growing from the crista petrosa of the fangs; but the term should be confined to tumours of new formation that consist of one or more of the dental tissues. Of these there are three varieties: (1) Enamel plates situated usually about the neck of the tooth. They are small, and look like pearly drops of congealed wax. They give rise to no symptoms. (2) Dentine tubercles projecting into the pulp cavity. They are only recognised after extraction for neuralgia. (3) "Warty teeth" or "dentinal odontomes" (Broca). These are the most important of dental tumours. They grow from the neck or fang, and form lobulated masses embedded in the jaw, which they absorb by their pressure; or they project from the teeth sockets. Billroth figures one more than an inch in diameter. Their structure is made up of one or more of the tooth elements. Usually there is an admixture of bone with irregularly disposed dentine or enamel.

A more recent classification of the odontomata is that based upon the portion of tooth-germ or tooth from which the tumour originates; consequently dentigerous cysts and the multilocular cystic tumour of Eve are included. The following description is taken mainly from Bland Sutton's work on "Tumours."

1. Epithelial odontome : from the enamel organ.
2. Follicular odontome
3. Fibrous odontome
4. Cementome
5. Compound follicular odontome
6. Radicular odontome : from the papilla.
7. Composite odontome : from the whole germ.

} from the tooth-follicle.



Fig. 80.—Dentigerous Cyst of Lower Jaw.  
(Follicular Odontome.)

The left side of the body of the lower jaw expanded with a large cyst, to the inner wall of which a retained canine tooth is attached. The cyst was lined with a thick vascular membrane composed of granulation and fibrous tissue, and showing no trace of epithelium. From a girl aged 13. (No. 2195 Royal College of Surgeons' Museum.) *a*, Molar tooth; *b*, tooth in cyst; *c*, coronoid process; *d*, condyle of jaw; *e*, cavity of cyst.

1. Epithelial odontome — multilocular cystic tumour of Eve —cystic epithelioma of jaw. These probably arise from persistent portions of the epithelium of the enamel organ. (Figs. 10, 81.)

2. Follicular odontomes — dentigerous cysts. The tumour consists of an expanded tooth-follicle. It may attain a large size. The cyst contains mucoid fluid and some part of an imperfectly developed tooth (Fig. 80).

3. Fibrous odontomes are excessive overgrowths of the tooth-sac. They consist of laminae of fibrous tissue, more or less calcified.

4. Cementomes are really ossified thickened capsules.

5. Compound follicular odontomes. "If the thickened capsule ossifies sporadically instead of *en masse* a curious condition is brought about, for the tumour will then contain a number of small teeth or denticles, consisting of cementum, or dentine, or even ill-shaped teeth, composed of three dental elements: cementum, dentine, and enamel. The number of teeth and denticles in such tumours varies greatly, and may reach a total of three or four hundred." (Sutton.)

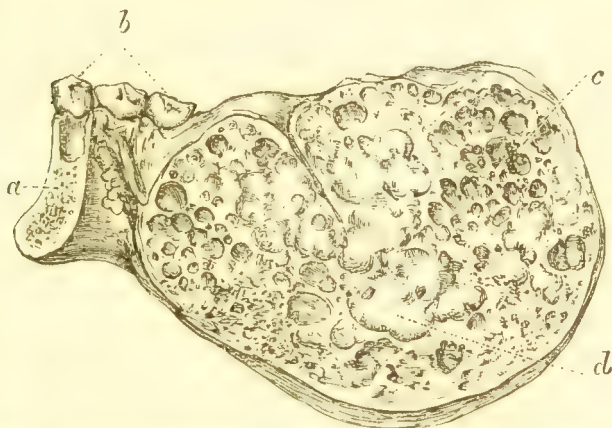


Fig. 81.—Cystic Epithelioma (Epithelial Odontome) of the Lower Jaw.

*a*, Divided bone; *b*, teeth; *c*, cysts containing a glairy fluid; *d*, lobules of the growth. For microscopical characters see Fig. 10. (St. Mary's Hospital Museum, No. 389.)

6. Radicular odontomes are formed after the crown of the tooth is completely developed, and therefore contain no enamel. They are composed of cement and dentine.

**Osteoid tumour.**—Virchow has described a new formation, the fundamental structure of which is identical with the "osteoid tissue" found beneath the periosteum in rickets. (*Vide* Rickets, page 181.) These growths are smooth or lobulated. They may attain considerable size. Clinically they are more nearly related



to the sarcomata than to the osteomata, for they generalise in the internal organs. They consist of spongy bone tissue, with an uncertain amount of embryonic, fibrous, or cartilaginous material; hence they may, by the unaided eye, be mistaken for tumours composed of one or other of these last-named tissues.

*Microscopy.* — Trabeculae of various sizes and shapes alternate with layers of connective tissue. They consist of a homogeneous or faintly fibrillated matrix in which angular corpuscles are embedded. These corpuscles have only short processes, and do not form a lacunar inosculating system as in true bone, although the spaces in which they lie are said to join minute channels in the intertrabecular tissue. The matrix is often calcified in patches, or it may be in its entirety. The fibrous tissue between the trabeculae carries the blood-vessels for the support and growth of the tumour. In it islets of cartilage are distributed, and when in large amount the name "osteoid chondroma" is given to it. According to Virchow, the osteoid tissue is not confined to new growths and rickets, but is met with in a minor degree beneath the periosteum during the normal development of bone. If this be so, it goes to show that the tumours under consideration are built upon the type of embryonic tissue of a specialised form, and to confirm the view of their histological relationship with the sarcomata, which in our opinion they really are.

## CHAPTER XCI.

## THE MYXOMATA.

WHEN treating of mucoid degeneration it was pointed out that certain tumours, especially the sarcomata, were liable to undergo this change; and that it lay at the foundation of true cystic development. (*Vide* Fig. 69.) But the term myxoma indicates those growths in which the embryonic tissue necessarily passes to the next phase of organisation, or that between indifferent cell-growth and fully formed connective tissue.

Their physiological type occurs widely distributed in the fœtus, and in the permanent mucous tissue of the vitreous humour and umbilical cord. The pure myxomata are generally benign, having but little tendency to recur after complete removal, or to generalise in distant parts.

This does not hold good concerning some mixed tumours, of which mucous tissue is a constituent—*e.g.* myxo-sarcoma. The gravity of these cases depends upon the more malignant element and the proportion it bears to the whole tumour. And there is this to be said, that a myxoma, representing as it does developmental tissue, is more likely than other simple tumours to revert to the embryonic type and become malignant. The association may be manifested quite early, or it may only show itself after a long interval.

To the naked eye the myxomata appear as semi-translucent gelatinous masses, of pretty uniform consistence and colour, unless perchance they be varied by patches of hæmorrhage, and softening from fatty degeneration and liquefaction of the intercellular substance. A glairy glutinous fluid is obtained by



scraping ; this consists of mucin mixed with cells of the growth, and blood-corpuscles. It is very different from the lactescent juice of cancers. The greater number grow from the mucous membranes in the form of *polypi*, or from connective tissue. They are also found in the voluntary muscles. According to Virchow, *hydatid moles* of the placenta are of the same nature. These moniliform growths start from the chorionic villi, which consist normally of mucous tissue. Many of the so-called *neuromata* are really myxomata, springing from the connective tissue of the nerves, the fibres of which are usually spread over the surface of the tumour, though occasionally they pass through the centre. There may be one or more ; when multiple they are distributed over the branches of a nerve or nerve plexus. They are exceedingly painful from pressure on the nerve-fibres.

Similar growths implicate the nerve centres. We have met with them both in the brain and spinal cord, arising apparently from the membranes.

The myxomata are chiefly found in early life, especially the polypoid variety.

**Nasal mucous polypi** are nearly always multiple. They are attached almost exclusively to the outer walls of the nasal fossæ. In aspect they are grey and glistening. A thin watery mucus exudes from their surface. They differ from the succulent fleshy "nasopharyngeal polypi" as follows : (1) They grow from the mucous membrane and not from the periosteum ; (2) their pedicles are very narrow, often filamentous ; (3) when completely removed they do not recur ; (4) they do not absorb and destroy neighbouring structures. Though one operation may not suffice for a cure, it does not point to a "recurrence" in the ordinary acceptation of the term, but to the existence of other polypi too small to be grasped by the forceps.

**Microscopy.**—All possess a homogeneous gelatinous intercellular substance. The cells embedded in this are round and oval in some specimens; stellate, branched, anastomotic in others, the outrunners forming a delicate plexus of fibres throughout the growth. But in by far the greater majority the cells are polymorphous, round, oval, stellate in varying



Fig. 82.—Mucous Polypus of the Bladder.

*a*, Squamous epithelium; *b*, blood-vessels; *c*, stellate corpuscles *d*, nucleus of corpuscle.  $\times 300$ . (See Fig. 69.)

proportions. This is well seen in Fig. 82, taken from a rare specimen of mucous polypus of the bladder (Fig. 69). Unless the section be stained the cells are barely visible, since their refractive index is nearly that of the ground substance. The myxomata growing in connective tissue have, as a rule, a thin capsule. Mucous polypi are covered with epithelium, like that of the region in which they are found. Thus, in

the bladder the superficial cells at least are flattened. In the nose they are columnar and ciliated.

The blood-vessels are easily observed. They have distinct walls, but, the lateral support being unstable, they easily rupture, hence the frequency of capillary hæmorrhages into the growth.

Elastic fibres and fat cells are sometimes present, so are cysts. Occasionally in mucous polypi glandular prolongations can be traced from the surface inwards. In them, too, subject as they are to physical injury, nutritive modifications are far from rare—hæmorrhage, inflammation, and gangrene from bruising or twisting of their pedicles.

## CHAPTER XCII.

## THE NEUROMATA.

Most of the tumours designated neuromata are either fibromata or myxomata growing from the connective tissue of the nerves. True neuromata are always homologous, and never generalise, although they may be multiple. They contain nerve fibres of new formation. There are two varieties, according as they contain medullated or non-medullated fibres, the myeline and amyeline neuromata of Virchow. Nerve cells are only met with in the tumours springing from the cerebral or spinal centres, and in some dermoid cysts of the ovary.

**Medullated or fascicular neuromata** are much more common than the non-medullated, but both are rare. The best example is seen in the clubbed extremities of the nerves in amputation stumps. In the regeneration of divided or wounded nerves new fibres are developed either from the elongation of the proximal ends of the severed fibres or from the conversion of the fusiform cells of the granulation tissue. The latter is more probable, as it explains the mode of formation of cicatricial neuromata. Even here the greater part of the enlargement consists of dense fibrous tissue. On strict pathological grounds these bulbous ends of nerves should be relegated to the inflammatory new formations. A similar development of nerve fibres is now and then met with in the continuity of nerve trunks, where it gives rise to fusiform swellings. Medullated nerve fibres are occasionally met with in dermoid cysts.

**Non-medullated or amyeline neuromata** are described by Virchow as occurring in connection

with the brain and spinal cord. They contain Remak's fibres, neuroglia, and nerve cells. The isolated patches of nerve tissue found in some congenital encephaloceles are the remains of herniated brain substance, and not new formations. The bulgings of the spinal cord on division of the membranes must not be mistaken for neuromata.

Non-medullated nerve-fibres are developed in some dermoid cysts.

The so-called neuromata of the optic nerve are either mucous or gliomatous tumours.

## CHAPTER XCIII.

## THE MYOMATA.

TUMOURS composed of muscular tissue alone are very rare, but complex growths of which it is a constituent are more common.

There are two varieties, striped and unstriped.

1. **Myomata with smooth fibres.** — These are always found in connection with the muscles of organic life. The best examples are those growing from the uterus and alimentary canal. In these situations they are either embedded in the muscular walls, or project beneath the mucous or serous membrane in the form of polypi. The submucous and subperitoneal myomata are probably intramural in their origin and are forced to the surface by the contraction of the muscles in which they are developed.

*Uterine* myomata are often very large, filling and distending the cavity of the uterus, or occupying the pelvis and abdomen external to that organ. Fibrous tissue enters largely into their composition, and eventually may so exceed the muscular elements as apparently to obliterate them. On this account some pathologists class these tumours with the fibromata, or notify their doubtful nature by the term “fibroid.” They are more correctly described as fibro-myomata or myo-fibromata (Fig. 83). The muscular fibre cells can be dissociated from the connective tissue by dissolving the latter in a 20 per cent. solution of nitric acid.

These myomata are smooth or lobulated. On section they are seen to consist of interlacing bundles of fibres, which often form concentric nodules; connective tissue carrying blood-vessels fills up the



intervals between the muscular fasciculi. They are generally circumscribed, but may be diffuse. They are for the most part very firm, and creak on being cut with a knife; but a few are more succulent, and contain large dilated vessels.

In the œsophagus, stomach, and intestine unstriped myomata are occasionally seen as small interstitial nodules or submucous polypi. Like the uterine growths, they may be single or multiple.

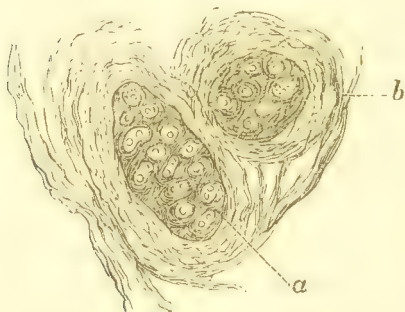


Fig. 83.—Myo-Fibroma of Uterus.

Transverse section of bundle of muscular fibres embedded in connective tissue, *b*. The tumour was as hard as cartilage.  $\times 265$ .

Muscular fibre is largely developed in the prostate glands of old men, as part of a diffuse hypertrophy, or as a circumscribed tumour. (*Vide* p. 449.)

*Nutritive modifications.*—(1) Calcification is very common, especially in “uterine fibroids.” The author once saw a completely

petrified uterine myo-fibroma. It was as large as a cricket ball, and was attached to the uterus by a fibrous pedicle a foot long. (By the twisting of such pedicle the vessels supplying the tumour may be strangulated.) (2) *Fatty degeneration* usually accompanies calcification. (3) *Mucoid* softening occurs sometimes, and gives rise to regular cysts or large anfractuous cavities.

Clinically, these tumours are benign, but they may be serious on account of the hæmorrhage they occasion and the mechanical interference with the functions of organs. In one case a uterine fibroid caused fatal obstruction of the bowels by clamping the rectum against the brim of the true pelvis.

**2. Myomata with striped fibres.** These are infinitely rarer than the preceding. They are seldom, if ever, found as simple tumours. Striped muscular fibres enter into the composition of some complex cystic formations in the ovary, and of some congenital tumours of other parts. The author was shown a sarcoma of the kidney by Mr. W. Pye, in which they were beautifully developed.

The unstriped myomata are homologous in their origin, the striped variety usually heterologous.

## CHAPTER XCIV.

## THE ANGIOMATA.

TYPE : blood-vessels.—They must be distinguished from aneurisms and varices, which are only dilatations of pre-existing vessels. It is doubtful whether aneurism by anastomosis should be included in this group; probably not, for there is want of proof that they consist of other than general and varicose dilatations.

There are two species of angioma : (1) simple, (2) cavernous.

(1) **Simple angiomata; telangiectases; congenital nævi.**—These affect the skin and subcutaneous tissue. They appear at or soon after birth, and increase by new formation of vessels. In colour they vary, being scarlet, purple, or blue, according to the proportion severally of arteries, capillaries, and veins that enter into their composition; and to the rate of the blood current through them. They are most common about the face and neck. They form soft spongy swellings, embedded in the skin, or raised from the surface as broad sessile elevations. They are composed of a number of flexuous intertwining vessels, held together by a scanty amount of areolar tissue. The vessels for the most part consist of capillaries, marked here and there with lateral loops and ampullary dilatations. Their walls, which are very delicate, are constructed of elongated cells. Muscular fibres are developed in some of the older and larger vessels; in fact, there may be well-formed arteries and veins. Occasionally they become obsolescent, being converted into patches of cicatricial tissue.

(2) **Cavernous angiomata; venous nævi; erectile tumours** (Dupuytren).—They are situated chiefly in and beneath the skin, but they are also found in the orbit and in the internal organs—*e.g.* the liver (Virchow). On the cutaneous surface they appear bluish. They can be greatly reduced in size by pressure. They very rarely pulsate. In children



Fig. 84.—Subcutaneous Venous Nævus  
a, Fibrous stroma; b, vascular canal.  $\times 200$ .

fat often enters largely into their composition (nævoid lipomata).

The vessels form a system of wide intersecting canals, alternating with trabeculæ of connective tissue, reminding one of the alveolar arrangement in carcinoma. They are lined with fusiform endothelial cells. Their walls consist of the fibrous tissue of the stroma, and sometimes contain muscular fibres and fat cells, but there are no regularly arranged tunics. The stroma is said to possess vasa vasorum and even nerves (Esmarch), but the latter are probably the remains of the normal tissue invaded by the growth. The blood spaces show a natural injection (*vide* Fig. 84)

in sections hardened by alcohol. The cavernous system is formed by dilatation of the vessels and by absorption of their walls. Where development is active the vessels are small as compared with the width of the intervening connective-tissue trabeculae, but subsequently this disparity is reversed. Rindfleisch explains it by assuming that of the two sets of intersecting columns (in this case represented by blood and connective tissue respectively), if one shrinks the other becomes enlarged. But any contraction of the stroma must be interstitial, and pretty equal in all directions, and therefore cause a diminution in the calibre of the vessels; besides, in cirrhosis of the liver, and in the cicatrisation of wounds, many of the capillaries are strangulated and finally obliterated. It seems more probable, then, that the enlargement of the vessels in angioma takes place at the expense of the stroma, and that the atrophy of the latter is due to the pressure of the blood in the cavernous spaces.

New vessels are formed by caecal protrusions from those already in existence. It is possible that some are developed from formative cells, as in the organisation of blood clots and inflammatory exudations, and that later they join one another and the vessels of the initial growth.

When the vessels increase rapidly in size and number they are surrounded by a richly corpuscular tissue, and the growth may then simulate a sarcoma. On the other hand, vascular pulsatile sarcomatous tumours of bone have been erroneously considered as "aneurisms" or *angiomata*.

**Secondary changes.**—(1) Calcification occurs now and then in the walls of cavernous naevi; (2) cysts may form by the expansion of the walls of one or more of the vessels after the latter have been strangulated, and occluded by coagula. The contents

of such cysts consist of liquefied clot and serous exudation (E. Wagner).

(3) The tumour may cease to grow, and shrink to a fleshy fibrous mass, "degenerated nævus"; this is chiefly seen in the scalp.

It may be noted that "vascular tumour of the meatus urinarius" (caruncle) is nævoid in its nature.

Mr. Barker has described a case of cavernous nævus of the rectum.\*

*Vide* Trans. Med.-Chir. Soc., vol. lvi. p. 229; 1883.



## CHAPTER XCV.

## THE SARCOMATA.

THE **sarcomata** are tumours that have their type in embryonic connective tissue. The simplest form is that consisting of an aggregation of round cells held together by a scanty homogeneous substance and traversed by capillary blood-vessels. It is impossible, from a microscopical examination alone, to tell it from inflammatory granulation tissue; in fact, it is sometimes called "granulation sarcoma." More frequently, however, the growth is not so primitive in structure; the cells enlarge and elongate, becoming spindle-shaped; or fibrous tissue is found more or less abundantly; or nodules of cartilage spring up here and there, and so on, until the most diversified group of tumours is completed.

Whatever modification they undergo, with a few rare exceptions, they do not assume that regular alveolar arrangement so characteristic of cancers. Having their type in embryonic tissue, they tend to follow it in the same lines of development; but they differ from it in that they never reach finality of growth, for there is no end to their formative activity.

The **cells** of which they are chiefly composed vary much in size and shape. Most commonly they are round or fusiform; but in the central sarcomata of bone large numbers are naked masses of protoplasm, with, it may be, as many as twenty nuclei.

They may become so attenuated that when closely packed they appear at first sight like bundles of fibrous tissue.

Various-shaped cells are often found in the same section.

The *nutritive modifications* are more numerous than in any other class of tumours; *e.g.* *melanosis* is much more common than in cancers. *Mucoid degeneration* accounts for the frequency of essential cysts—essential inasmuch as many arise, not from accidental rupture of vessels, nor from liquefaction by fatty degeneration from deficiency of nutriment, but from certain cells set apart for this inherent transformation.

The **intercellular substance** varies much in amount and form. In some cases (*e.g.* the round and large spindle-celled varieties) the cells appear in actual contact; in others, composed mainly of small spindle cells, the matrix is often very abundant, and is homogeneous or fibrillated. In lympho-sarcoma there is a delicate reticulum of fibres between the cells, resembling the stroma of a lymphatic gland; and in glioma of the nerve centres and retina a similar arrangement is found.

It is subject to calcification and other changes.

**Blood-vessels** traverse the tumour in all directions. Their walls are very thin, and are constructed by the cells of the growth, elongated, perhaps, and arranged end to end, but never forming distinct coats. They readily dilate and rupture; hence aneurismal pouches and hæmorrhages.

From the frequent admixture of other than embryonic elements, sarcomata form composite tumours; the terms myxo-sarcoma, fibro-sarcoma, and chondro-sarcoma explain themselves.

Although any variety may be found in any tissue, there is still a well-marked tendency in many instances to affect certain structures; thus the pigmented form is mostly found in the choroid and skin; the cystic, in the bones, testicles, and breast; the myeloid, in cancellous bone; the round, mixed, and spindle-celled growing from bones and fasciæ.

Sarcomata destroy the tissues in which they grow in three ways: (1) By direct pressure, causing atrophy in the same way as an aneurism; (2) by infiltration; (3) by compression or invasion of large vessels.

When the growth in a bone is central, we often find expansion with the wasting, which accounts for the "egg shell" crackling over such tumours, especially in the jaws; and for some of the so-called spontaneous fractures of the long bones.

When pulsatile, and situated in the regions of large arteries—*e.g.* the popliteal space or the pelvis—it may be difficult to diagnose them from aneurisms, for they give a distinct bruit and thrill; the latter signs, however, are more uniformly diffused in sarcoma, and the murmur is not conducted along the main artery; again, they cannot be so easily emptied by pressure. They do not refill so suddenly as aneurisms.

The pulsation is increased by the formation of aneurismal dilations of the vessels, and hence such sarcomata of bone are often described as "*aneurisms of bone.*"

Sarcomata springing from the surface of bones often present a radiating appearance (Fig. 37) from the centrifugal direction of the blood pressure, and when ossification occurs the new bone often follows the same lines in the form of delicate feathery sprays.

**Varieties of sarcoma.** — These are based, firstly, upon the *shape of the cells* that predominate in the tumour (round-celled, spindle-celled, myeloid); secondly, upon some *modification of structure*, either in the arrangement of the cells and disposition of the intercellular substance (lympho-sarcoma, alveolar sarcoma), or from admixture with other than embryonic tissue (chondro-sarcoma, osteo-sarcoma); thirdly, upon some *nutritive change* (melanotic sarcoma, cystic sarcoma).

As to relative malignancy, round-celled sarcoma takes the lead, for the whole of the vital energy of the tumour is expended in growth, to the exclusion of a higher development. Next comes the large spindle-celled variety, the cells of which are closely packed, there being but little ground substance. Then, in order, are the mixed round and spindle-celled, the small spindle-celled, and the myeloid or giant-celled. The melanotic and alveolar are, as a rule, very malignant.

Chondrifying and ossifying sarcomata are malignant in proportion to the amount of embryonic tissue they contain.

(1) **Round-celled sarcoma, embryo-plastic tumour** (Lebert), **encephaloid sarcoma** (Cornil and Ranvier).—Of this there are two varieties, small- and large-celled. They are of very rapid growth, often ending fatally in a few months, especially in children, the formative activity of whose tissues is very great.

They are of soft, brain-like consistence, of a pinkish-grey or yellowish-white colour. The walls of the vessels, being very thin and badly supported, rupture spontaneously, or on the slightest pressure, giving rise to patches of extravasation, or even large blood cysts. Having gained the surface, they form the so-called “fungus hamatodes,” from their rapidity of growth and tendency to bleed (the same name is applied to fungating encephaloid cancer). The multiplication of the cells outstripping the formation of vessels for their support, and the acute interruption of nutrition from rupture of capillaries, lead to fatty degeneration and death of the cells, the softened débris mixed with blood forming a grumous pultaceous mass. Their elasticity may easily be mistaken for the fluctuation of an abscess, but on passing a trochar little but blood escapes. They infiltrate the surrounding tissues, so that removal is difficult and

recurrence rapid and certain. They generalise in the internal organs, in the lymphatic glands, and even in bone; but death may supervene so shortly that there may not be time for secondary growths to appear. They affect almost all the tissues, but the skin, bones, and fasciæ are their seats of election. Microscopically, they consist for the most part of simple embryonic cells embedded in a scanty homogeneous or granular matrix. Sometimes the cells are more voluminous, with large bright nuclei.

(2) **Spindle-celled sarcoma.**—Of this there are two varieties, the large- and the small-celled. The *large spindle-celled* consists of long, tapering cells (Fig. 85), closely packed in a scanty homogeneous substance. The cells contain one or more oval nuclei and nucleoli. As they show little or no tendency to form fibrous tissue, they are much softer and more malignant than the small-celled variety. The small spindle-celled sarcomata include the “fibroplastic tumour” of Lebert and the “recurrent fibroid” of Paget. The relative amount of cells and intercellular substance varies greatly; in some the cells constitute the main part of the tumour; in others they are embedded in a large quantity of ground material, homogeneous, granular, or fibrillated. Their appearance on section, rate of growth, and malignancy will thus differ within very wide limits; they may run closely the large-celled variety in gravity, or they may approach the simple fibromata. It may be here noted that the unstriped myomata and the fibromata in the early stages of their growth contain fusiform cells which cannot be distinguished from those of sarcomata; we must then turn to other portions of the same specimen, or wait for the further development of the tumour, to decide upon its real nature.

The majority of small spindle-celled sarcomata are firm in consistence, generally encapsuled, and thus

non-infiltrating. On section they appear of a greyish-white colour, and are seen to be traversed by intersecting bands of what appears like fibrous tissue. However hard, they do not retract and cup in the centre like scirrhus cancer; for their density is more uniform throughout. They grow from the bones, fasciæ, muscles, and connective tissue generally. When they recur after removal, successive growths are liable to be softer and more cellular

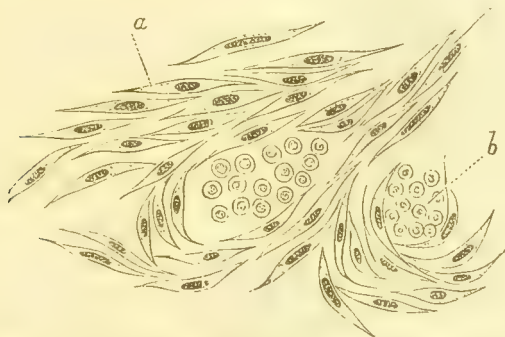


Fig. 85.—Large Spindle-celled Sarcoma from the Fascia Lata of the Thigh.

*a*, Spindle cells with fusiform nuclei; *b*, bundle of spindle cells in transverse section.  $\times 265$ .

than their predecessors, so that it is not uncommon for the intervals of immunity to get shorter and shorter. They generalise in the internal organs, but the lymphatic glands are rarely affected. After several years, death may ensue from exhaustion from operations and ulceration of the tumour, and not a secondary growth be found. Finally, the cure may be complete after one or more removals.

They may ossify or calcify, more frequently the latter. If the lime salts be dissolved out, the true structure becomes manifest on section; cells which were before hidden by the petrification are brought into view.



(3) **Mixed round and spindle-celled sarcomata** are very common. Their characters depend to a great extent on the proportion of round to spindle cells.

(4) **Myeloid, or giant-celled sarcomata**, with

very few exceptions, grow from bone, and that too from the cancellous tissue. Their favourite seats are the jaws and the ends of the long bones, especially the upper end of the tibia and lower end of the femur, radius, and ulna (Fig. 86).

In the jaws they are either central, or they project from the sockets of the teeth as *epulides*. When central the pressure of the growth causes atrophy of the compact laminae; but this is partly compensated by the deposit of new bone on the outside from the irritation of the periosteum. This continuous absorp-

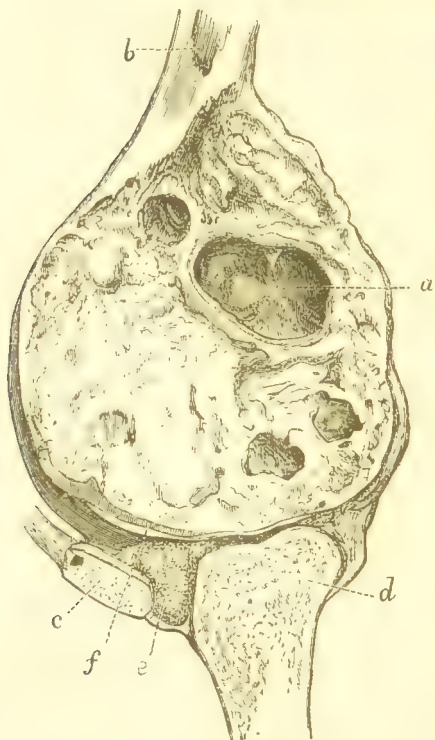


Fig. 86.—Cystic Myeloid Sarcoma of the lower end of the Femur.

*a*, Cyst; *b*, medullary canal of the femur; *c*, patella; *d*, tibia; *e*, fat beneath the synovial membrane of the knee-joint; *f*, remains of the femoral articular cartilage.

tion and deposit accounts for the hollow perforated shells sometimes seen after maceration.

Their growth is slow, even when the skin or mucous membrane has ulcerated over them. Complete

removal often effects a cure ; very rarely do they give rise to secondary deposits. They are often cystic, seldom extensively ossified. To the naked eye they appear reddish-brown, or ochre-coloured, or pale yellowish-white, with splashes of a ruddier tint. Their uniform firmness is broken by patches of ossification and fatty degeneration, or by cysts developing, or fully formed. When central their outline is regular,

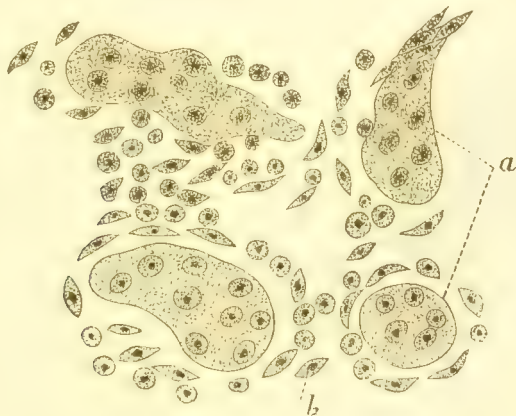


Fig. 87.—Myeloid Epulis from Lower Jaw.  
*a*, Multinucleated giant cells ; *b*, oval cell.  $\times 265$ .

and they may appear encapsuled (Fig. 86) ; when springing from a surface, lobulation is not uncommon.

The essential structure consists of large, round, oval, or branched cells, with many nuclei embedded in homogeneous or granular protoplasm. The nuclei are generally scattered, and thus contrast strongly with those of the giant cells of tubercle, which show a great tendency to circumferential arrangement. These giant cells, which are from  $\frac{1}{300}$  to  $\frac{1}{1000}$  inch in diameter, are like those of foetal marrow, hence the favourite situation of these tumours, and their name, myeloid. They may be sparsely scattered in a bed of round and spindle cells (Fig. 87). They may

constitute the greater part of the tumour. When found in peripheral sarcoma of bone, which is comparatively rare, they do not confer any clinical significance.

*Subvarieties of sarcoma.*—1. *Lymphosarcoma*, resembling the structure of a lymphatic gland, is really a modification of the round-celled variety. There



Fig. 88.—Alveolar Sarcoma of Ilium.

*a*, Stroma composed of branched cells; *b*, round nucleated cells filling alveoli.  $\times 265$ .

is a delicate reticulum, in the meshes of which the cells are enclosed; this is found in the malignant lymphadenomata, and in some sarcomata of bone, especially in children.

2. *Alveolar sarcoma* is chiefly found in bone, muscle, and skin. It frequently starts from moles. It seems to form an anatomical link between sarcoma and cancer; but its

affinities, structural and clinical, cling to the former. It differs from cancer (*a*) in that the fibres, which are often only the outrunners of cells, ramify between the individual cells as well as between the groups; (*b*) in that the cells are on the connective-tissue type, and are less easily removed from the stroma than in cancer; (*c*) the alveolar retiform arrangement is more uniform (Fig. 88).

3. *Glioma*, *gliosarcoma*, attacks the nerve centres, nerves, and retina (Fig. 90); in the latter case it may be congenital, and it usually occurs in early childhood. It

is developed after the manner of the neuroglia or connective tissue of the nerve centres. A typical section shows a delicate reticulum between the cells, but often only a scanty homogeneous matrix exists (Fig. 89). The cells, mostly round, like lymph cells, are occasionally fusiform. It is subject to fatty and mucoid degeneration. When it grows from the retina, the fundus oculi presents a lustrous bright-yellowish



Fig. 89.—Glioma of the Retina.

*a*, Cells of the growth, the greater number of which are round. The rest are out-shaped or stellate. The intercellular substance is faintly fibrillated. *b*, Blood-vessels. Their walls are composed of fusiform gliomatous cells.  $\times 265$ .

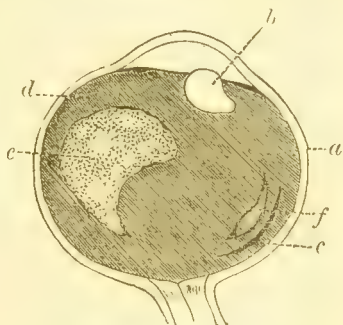


Fig. 90.—Glioma of the Retina.

*a*, Sclerotic; *b*, dislocated crystalline lens (it was quite transparent); *c*, remains of choroid; *d*, gelatinous mass of glioma; *e*, patch of cheesy-looking matter (fatty degeneration); *f*, cleft containing serous fluid. (Natural size.)

appearance, quite characteristic. It destroys all the structures, perforates the tunics, and forms a bleeding fungus.

If removed early it may not return; but recurrence in the optic nerve is to be feared.

4. *Melanotic sarcoma*.—Melanosis is more common in sarcoma than cancer, and of the former the spindle-celled variety is its chief seat. These sarcomata are prone to occur where pigment is normally present; hence the choroid, iris, and the skin are the structures usually affected; though they may be found in muscle, lymphatic glands, and other tissues.

The pigment is in the form of minute granules. It occupies chiefly the cells ; probably that seen in the matrix is merely the remnant of disintegrated cells. The granules are black from the first, and thus differ from those formed from extravasated blood (Cornil and Ranvier). The deposit commences around the nuclei, which for some time by their brightness contrast strongly with the dull, dark colour of the pigment. The amount of pigmentation varies in different tumours, and in different cells of the same tumour, so that to the naked eye these growths appear grey, sepia-coloured, or jet black. Secondary growths present the same characters. Inasmuch as these tumours are mostly round or large spindle-celled, the prognosis is grave. The tendency to secondary deposit in the associated lymphatic glands is very marked.

5. *Mucous sarcoma*. — With the exception of growths essentially cystic, sarcomata are more subject to the formation of *cysts* than any other group of tumours. We have said that cavities arising from extravasations of blood and fatty degeneration are pathological accidents ; but a sarcoma may be honey-combed by regular spaces with definite walls, and filled with gelatinous matter derived from mucoid degeneration. The coalescence of these cysts may so hollow out the tumour that but little solid matter is left except the septa and marginal portion, which show the change in progress. Fresh growths encroaching upon these spaces project into the interior, and give rise to the so-called *intracystic tumours* ; and by the junction of papillary processes of the latter, secondary cysts are formed between them. The contents of the cysts are usually mucoid, but sometimes the fluid is clear and limpid, and then perhaps has been derived from serous exudation between the lobules and the delicate capsules covering them ; and it may be coloured with blood derived from capillary



rupture. Cystic sarcoma in the popliteal space may be easily mistaken for an enlarged bursa.\*

6. *Ossifying sarcoma*.—Osteosarcoma is found chiefly in the jaws and at the ends of the long bones. The bone may be deposited in isolated patches, but more commonly it occurs as a radiating or nodular outgrowth from the base of the tumour, as in subperiosteal epiphyseal sarcomata, and some epulides (Fig. 37). The cells of the tumour are converted into bone corpuscles with long processes. Haversian canals and canaliculi are constructed, but lamellation is rare.

It may be impossible by the unaided eye to tell an ossifying from a calcifying sarcoma.

7. *Chondrosarcoma* by preference attacks the ends of the long bones and the testicles. I once amputated at the hip-joint for a cystic chondrosarcoma involving the whole length of the shaft of the femur; it stopped short at the level of the epiphyseal cartilages.

These tumours are by some placed with the enchondromata, but it is better to name them generically from the more malignant constituent. Between the nodules of cartilage are round and spindle cells. They often ossify and calcify.

When the islets of sarcoma cells are embedded in mucous tissue the growth is termed *myxosarcoma*. Surface sarcomata, whether growing from the skin or into cavities (*e.g.* the maxillary antrum), are habitually *papillated*.

The cells of a sarcoma may be filled with globules of fat, not derived from degeneration, but from simple infiltration, *lipomatous sarcoma*. Contrasting the central with the subperiosteal sarcomata of bone, Mr. Butlin, who has thoroughly studied the pathology of these tumours, says the former more often pulsate do not show a radiated structure, rarely ossify or chondrify, are found in older subjects, and are less

\* Vide *Lancet*, March 23, 1878, p. 418.



malignant than peripheral sarcomata, for they have a slower growth, and do not infiltrate the surrounding structure and lymphatic glands to the same extent.

*Multiplicity of growth* in sarcomata may be due : (1) To a common origin, especially when many bones are affected ; (2) to true dissemination ; (3) to infiltration of lymphatic glands by a continuous invasion from the primary growth.

The **psammomata**.—These are small tumours found in connection with the membranes of the brain. They are impregnated with calcareous salts, hence the name. In structure they are composed of large flat angular cells, arranged in concentric laminae. Virchow describes them as epithelial conglomerations, or nests, derived from the cells lining the *ependyma ventriculorum*. Cornil and Ranvier, who place them among the sarcomata, have traced their development from vascular buds of the choroid plexuses, and other parts of the pia mater. They explain the flattened laminated character of the cells by the pressure of the blood in the early stages of growth. If formed in this way they subsequently become isolated by obliteration of the vascular pedicles. They are of no clinical importance.

## CHAPTER XCVI.

## THE LYMPHADENOMATA.

THE term lymphadenoma or lymphoma is commonly applied to any hyperplastic enlargement of a lymphatic gland, whether it arises spontaneously, or from simple irritation. It is difficult to separate the two groups; for, in the first place, there is no reason why a peripheral irritation of the lymphatics should not excite the associated glands to renewed developmental activity; and, again, hypertrophy of the gland may continue long after the primary cause has disappeared, so that one is left in doubt as to its true nature. On the other hand, the enlargements due to injury lead to a typical end: subsidence, caseation, or suppuration (the two latter are pretty constant in tubercular subjects); whereas the non-inflammatory lymphomata tend to persist, affect other structures besides the glands, and very rarely suppurate.

The lymphomata may be arranged clinically under four heads: (1) The simple or benign lymphomata; (2) the malignant lymphomata, or lymphosarcomata of Virchow; (3) multiple lymphomata of the viscera (*e.g.* the liver, spleen, kidney, etc.), Hodgkin's disease, anæmia lymphatica, adénie (Trousseau); (4) the last-mentioned group associated with a marked increase in the number of white blood-corpuscles (leucæmia, leucocythæmia).

(1) The **benign lymphomata** are more numerous than the other varieties combined. They may or may not be traceable to injury or precedent disease. Rarely attaining a large size, they cease to grow after

a time, and remain quiescent, or shrink from fibroid condensation. They never extend beyond the glands involved, and are consequently homologous. They are the expression of tissue weakness, but are compatible with good general health. Their removal is not attended with any special danger. Usually only one gland or group of glands is affected. The neck is the seat of election; next to that the groin and axilla.

*General anatomy.*—The simple lymphomata are enclosed within a fibrous capsule, and retain the general outline of the lymphatic glands, being round, oval, or kidney-shaped. They are mostly firm, sometimes very hard, and the consistence is fairly uniform. On section they look greyish and semi-translucent, or dull white and opaque, according to the amount of fibrous tissue present. For the same reasons they are more or less homogeneous, or streaked with whitish trabeculæ.

*Microscopy.*—The ground substance looks glassy or fibrous, and forms meshes within which small uninucleated lymph cells are enclosed. In pencilled sections nuclei may be seen here and there at the nodal points of the reticulum.

(2) The **malignant lymphomata** usually involve several glands and sometimes several groups, cervical, mediastinal, abdominal, etc. Clinically, they are sarcomata. At first homologous, they may become heterologous by invasion of surrounding tissues. They are often lobulated from adhesion of contiguous growths. In preparations, the large vessels of the part are frequently seen to be embedded in the mass, or stretched over it. The elasticity of these tumours is so great that it may be mistaken for the fluctuation of an abscess. I have seen them incised on that supposition. Progressive anæmia complicates the disease, which is fatal from

asthenia, or pressure upon some vital organ. Multiplicity of growth does not imply dissemination, as in carcinoma; it is the sign of widespread instability of tissue in the lymphatic gland system. If left alone it is quite the exception for these tumours to fungate, but if incised they pursue an intractable course. Attempts at removal are often futile, and certainly dangerous, for during the operation the disease is rarely found to be as limited as appeared from a surface examination; besides, the patients are bad subjects for the repair of extensive injuries.

*General anatomy.*—The mass is bounded by a thin capsule, or, more rarely, it is infiltrating. In the latter case the original capsule of the gland has disappeared before the ravages of the disease. The cut surface appears grey or yellowish-grey throughout, or it is mottled and flecked with red from capillary extravasation. Patches of softening and caseation may be seen. The distinction between the medullary and cortical portions of the gland disappears.

*Microscopy.*—Small lymph cells form the greater part of the tumour; but there are also large multinucleated cells which indicate rapid growth, the nuclei having divided more quickly than the protoplasm. The stroma is very delicate; to see it the specimen must be shaken in water or lightly brushed. It is largely made up of branched cells.

(3) **Lymphomata of the viscera and other structures** are met with in the spleen, liver, kidneys, stomach, intestines, tonsils, and thymus gland. The lymphatic glands are usually involved at the same time. The liver and spleen attain a great magnitude; in one case the former weighed seven, and the latter eight pounds. The growth consists of an aggregation of nodules from the size of a pin's head to a marble. In the spleen it affects chiefly the Malpighian glomeruli; in the liver, the interaciinose tissue

(Fig. 91) ; in the stomach, the solitary glands ; in the intestine, Peyer's patches and the solitary glands. I have seen a lymphoma of the thymus as large as a cocoanut.

The minute structure is much the same as described in the other forms ; indeed, it may be said of all lymphomata that they are reproductions with variations of the adenoid tissue of His ; in some there is scarcely any departure from the normal ; in others the fibrous



Fig. 91.—Lymphoma of Liver, from a case of Leucocythæmia.

*a* Liver cells ; *b*, adenoid or retiform tissue, showing nodal enlargements of the meshwork. Most of the lymph cells have been removed.  $\times 265$ .

or the cellular element predominates, or the growth is modified by secondary nutritive changes. This adenoid tissue has a wide natural distribution, for, apart from the structures of which it is the chief constituent, it is met with in the liver, walls of the alimentary canal, around the bronchioles and arterioles of the lung, and the vessels of the nerve centres, and lastly in bone. The extensive range of distribution of the lymphomata as homologous growths is thus easily explained.

(4) **Leucocythæmia.**—In this disease there is a great increase in the number of white blood-corpuscles. It is associated with lymphoma of the spleen, or lymphatic glands, or both. Its ætiology is obscure, for,

as before said, the preceding group may exist without it. Again, it cannot be said that a morbidly increased formative activity of the lymphatic glandular system leads to an increased functional activity. During the course of the disease diffuse hæmorrhages may occur. This has to be borne in mind, to avoid mistaking the extravasations for abscesses of the subcutaneous, submucous, and intermuscular tissue.

#### THE LYMPHANGIOMATA.

Swellings consisting largely of distended lymphatics have been observed in the tongue (one form of hypertrophy of that organ), lips, cheek, and subcutaneous tissue of the abdomen, thigh, and genital organs. In some cases the dilatation and varicosity of the lymphatics have been traced into the neighbouring glands (adeno-lymphocele, Nelaton). In elephantiasis of the Arabs the newly-formed tissue contains numerous large lymph spaces filled with clear fluid; and this disease is known to be preceded and accompanied at intervals by lymphangitis, but whether there is any new formation of lymphatics is not certain. On anatomical grounds there is no reason why there should not be a new development in connection with the lymphatic, as well as with the blood vascular system.

The swellings above referred to are termed "cavernous lymphatic tumours," or lymphatic nævi. They form soft, doughy, or fluctuant masses, and occasionally exude lymph on the surface (lymphorrhœa).



## CHAPTER XCVII.

## THE PAPILLOMATA.

THE skin and mucous membranes, instead of forming a plane surface, are usually raised into papillary or rugose elevations, or involuted as sweat, sebaceous, or mucous glands. This disposition constitutes the basis of the papillomata and adenomata respectively. The new growth is not composed entirely of epithelium, for there is hypertrophy of the proper cutaneous and mucous tissues.

The **cutaneous papillomata** comprise warts (hard and soft), corns, and horny excrescences.

*Warts* are hypertrophic enlargements of the papillæ of the skin. In many cases they spring up spontaneously, but in others there is some precedent irritation. The former have a peculiar tendency to appear on the face and hands, especially in children: whilst the latter are prone to affect the skin at its junction with the mucous membrane, notably about the genital parts. *Gonorrhæal warts* are caused by friction and irritation from acrid discharges. They are inflammatory, but are classed with the papillomata on account of their structural identity, and because they often continue to grow long after the primary cause has been removed. They differ from syphilitic mucous tubercles in that they less rarely disappear spontaneously. They are usually multiple. When large they are known as *cauliflower excrescences*, which may attain the size of a cocoanut.

Warts on the general surface are either soft or hard. In each case there is overgrowth of the papillary and epidermal layers. The difference in density depends upon the amount of epithelium and

its degree of cornification. The soft variety is smoother than the hard, and is more often pigmented, and it has a greater tendency to affect persons advanced in age. Cutaneous warts consist simply of a group of enlarged papillæ, or the latter branched from division and subdivision, as seen in the scalp.

*Corns* are met with where the skin is subjected to intermittent pressure. The horny layer of the epidermis is very thick and dense, and not only covers the individual papillæ, but fills up the hollows between them, so that the surface is more or less smooth. The papillæ are atrophied in the centre from continuous pressure.

*Horny excrescences* are sometimes more than an inch in length. The hard epidermal cells are so firmly welded that there is little or no desquamation. They start either from the surface, or in the hair follicles and sebaceous glands. They have been found in dermoid ovarian cysts.

**Mucous papillomata** occur on all the mucous membranes, but they are more frequent where papillæ normally exist; *e.g.* in the tongue. They are more succulent than the cutaneous growths, and their density varies as the kind of epithelium. Thus, in the tongue, where it is squamous and stratified, they are firmer than in the rectum.

In the bladder in particular they form soft *villous growths*, with long processes, simple or branched; and here their vascularity is so great, and the vessels are so little supported by the surface epithelium, that profuse bleeding is not a rare event. Vesical papillomata are mostly seen near the orifices of the ureters, but the entire mucous membrane may be converted into a shaggy flocculent coating. The epithelium is squamous or columnar, and disposed in a single layer, or stratified (Fig. 92). In the rectum it is columnar. Papilloma of the larynx is nearly six times as common as

epithelioma. It is usually seated on or near the vocal cords.

**Papillomata of the serous membranes.—**

Some authors include hypertrophy of the synovial articular fringes in the papillomatous group. They



Fig. 92.—Simple Villous Tumour of the Bladder.

*a*, Columnar epithelial cells ; *b*, large capillary blood-vessels.  $\times 265$ .

have been described in the chapter on joint diseases (Fig. 49).

**Papillary forms of other growths.—**Most of the new formations arising from the skin and mucous membranes are prone to assume a papillary

surface outline. This is very marked in the malignant tumours, and especially in epithelioma; in fact, it is difficult in some cases to say with certainty whether a warty growth in its early stage is a papilloma or a papillary epithelioma. And, again, a papilloma may, by continued irritation, take on a cancerous nature.

From a mere surface view it is impossible to tell a villous tumour of the bladder from a villous cancer; but the former grows entirely from the surface, whereas the latter invades and infiltrates the entire thickness of the walls.

Sarcomata of the nasal fossa, antrum, and other cranial sinuses are often papillated. The intracystic formations of many ovarian tumours are foliated.

**General anatomy and histology.** — Where papillæ enter into the normal structure of the part, the type is simply maintained in the new growth. Thus in a wart the cutaneous papillæ are reproduced, though in a disorderly form; the basis of the tumour is made up of connective tissue rich in corpuscles; the vessels are plexiform or looped; the epidermal cells are more or less columnar below, squamous and stratified above; their extent, degree of cornification, and disposition vary in different cases, but they are always homologous. In epithelioma they are homologous at first, but afterwards essentially heterologous.

In mucous membranes devoid of papillæ the outline of the tumour can be explained by the looped arrangement of the vessels and the centrifugal direction of the vascular pressure, as in the granulations of an ulcer.

Under the microscope, transverse sections of the tips of the papillæ look very like the nests of epithelioma.

**Secondary changes.** — These are ulceration, hæmorrhage (surface and interstitial), pigmentation, epitheliomatous degeneration, and, in some cases, atrophy and complete obliteration.

Ulceration and hæmorrhage are rare in the cutaneous papillomata ; far from rare in the mucous, which are ill protected from friction and other sources of irritation. This is markedly the case with villous tumour of the bladder.

Pigmentation is almost confined to papilloma of the skin.

## CHAPTER XCVIII.

## ADENOID TUMOURS.

ANY new growth, simple or malignant, arising from glandular tissue has a tendency to assume the adenoid type. Adenoid tumours may be conveniently divided into four groups :

(1) *Malignant tumours*—*e.g.* scirrhus cancer of the breast. They are none the less malignant because

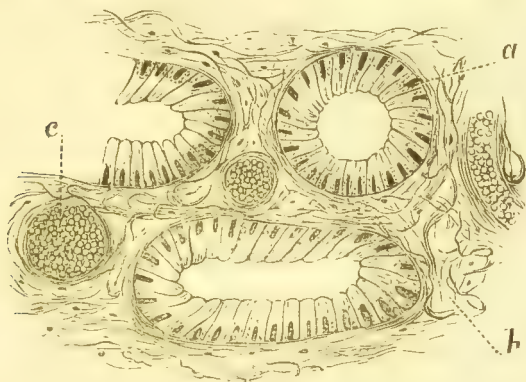


Fig. 93.—Fibro-glandular Polypus, from the Rectum of a Child.  
*a*, Acinus lined with columnar epithelium ; *b*, fibroma-nucleated stroma ; *c*, blood-vessel.  $\times 265$ .

stamped with the physiological likeness of the part affected.

(2) *Hypertrophies* of the thyroid, mammary, and prostate glands. In each there is considerable reproduction of glandular tissue. Their right to be classed with the adenoid growths is further shown by the fact that encapsuled masses, containing loculi with regularly-disposed epithelium, are also met with in these organs, either alone or in conjunction with the more diffused enlargement.



These are the cases that prove how difficult it sometimes is to distinguish between hypertrophy and tumour when no physiological reason can be assigned for the overgrowth, and when the microscopical characters are practically the same.

(3) The important group of tumours termed adenomata, or adenocoeles, of which the best instance

is seen in the breast (Fig. 94).

In it are included certain pedunculated tumours attached to the mucous surfaces — *e.g.* glandular polypus of the rectum. (*Vide* Fig. 93) In these the epithelial adenoid tissue forms acini, even in

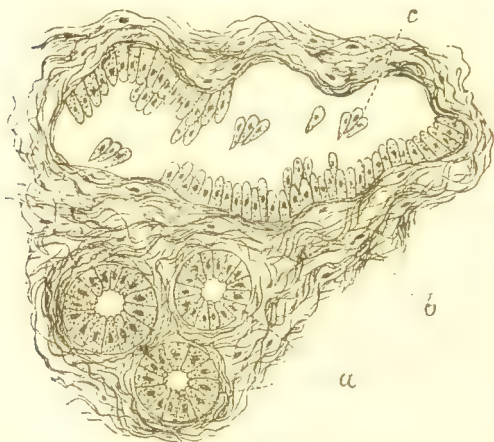


Fig. 94. Adenoma of the Breast.

*a*, Group of glandular ducts; *b*, fibrous stroma; *c*, cells broken away from their attachment.  $\times 265$ .

the deeper portions of the growth. This must not be confounded with simple involutions of the surface epithelium occasionally present in many polypi — *e.g.* mucous polypus of the nose.

(4) The lymphadenomata, which consist of tissue resembling that of a lymphatic gland.

#### **Adenoma, or adenocoele of the breast.**—

“Chronic mammary tumour” is met with at different ages, but chiefly in early and mid-adult life. It is either embedded in the gland or attached to the surface. In either case it is distinctly encapsuled, and, as a rule, freely movable apart from the breast.

It does not infect the lymphatic glands or internal

organs. It neither causes retraction of the nipple nor puckering of the skin. Ulceration is very rare, and this only happens when, by the size of the tumour, the skin has become atrophied by pressure, and exposed to injury. It is often lobulated. The density varies according to the amount of fibrous tissue present in the growth; it may be so hard as to simulate scirrhus. The size usually ranges from that of a marble to an orange, but there is no certainty. I have examined a specimen that weighed eight pounds. Unlike scirrhus, it does not cup on section, for the tension is fairly uniform throughout. The lobulation of the surface is continued into the interior; it may be scarcely visible, or so decided that the lobules appear partitioned off by fibrous dissepiments; it becomes more manifest on tearing portions of the growth from one another, for then the lobules "shell out." The lobules may be so small as to make the surface merely granular, or so large as to exceed a walnut in size. Scraping does not yield a milky juice, but a slightly turbid fluid, and coarse particles composed of cells and fibres, and often minute gland-buds. Fibrous tissue almost invariably exceeds in amount the glandular element. This has led to the designation *fibro-adenoma*.

*Microscopy.*—The *glandular spaces* show great diversity of outline, being round, oval, or sinuous. In extent, too, they vary much, being wide, like open ducts, or mere chinks in the fibrous stroma. Their interior is lined with columnar or cubical epithelium, usually in one layer, but sometimes the cells are two or three deep (Fig. 94). The central portion of each space appears clear, or granular from epithelial débris. There is a much closer resemblance to healthy gland tissue than in the alveoli of scirrhus cancer. The *stroma* is in most cases very dense, and its cells are sparsely scattered, but in some it is succulent and

richly corpuscular. In a series of cases one may notice a gradual declension from a firm, hard nodule of adeno-fibroma to a soft, rapidly enlarging adeno-sarcoma.

Cysts, which are by no means rare, arise for the most part from dilatation of the gland spaces: but some seem to be connected in their origin with the lymphatics, for I have found both cubical and pavement epithelium lining the interior. The fluid in the cysts is clear and mucoid, or opaque from admixture with blood, and then it may have a reddish-chocolate or sepia-like tint, according to the amount and age of the extravasation. In such cases any discoloration of the cyst wall and tissue around must not be mistaken for true pigmentation.

Now and then a considerable portion or even the whole of the breast is found to be enlarged and nodular on the surface, without any distinct capsular limitation to the growth. This has been termed "*diffuse adenoma*," or hypertrophy.

**Adenoma of the parotid** is met with in which the structure is entirely glandular, or fibro-glandular; but it is more often combined with mucous, cartilaginous, or embryonic tissue, one or more of them. There are two varieties, tubular and acinous. As in the case of the breast, it may be seated deeply in the gland, or loosely attached to the surface. In either case it starts beneath the capsule of the gland, though in its subsequent growth this may become so thinned as to be scarcely recognised, and this has led to the idea of the tumour being extracapsular from the beginning.

The submaxillary and sublingual glands are much less frequently affected.

**Adenoma of the prostate** is rarely met with as constituting the entire disease. Usually it forms part of a more general enlargement or hypertrophy.

Roundish masses, enclosed in more or less distinct capsules, project as pedunculated tumours into the bladder, or lie embedded in the substance of the gland. In the latter case they can sometimes be readily enucleated. The nodule from which Fig. 71 was taken was removed during the operation of lithotomy. It formed a bar to the passage of the stone, and was displaced by the forceps. It will be seen to consist of glandular and muscular elements in a nucleated fibrous stroma. The epithelium is columnar, as in the mucous glands of the prostate.

**Adenomata of the skin.**—Eve and Shattock have described adenomata of the skin which arise from the sebaceous glands. In Shattock's case the tumour formed a flattened, ovoidal, pedunculated mass, 3 centimètres in diameter.\*

The adenomata are perfectly benign, and the epithelial cells are homologous throughout. This at once distinguishes them from the carcinomata, where the epithelium, though at first homologous, subsequently becomes heterologous—*i.e.* it is found invading tissues from which it is normally absent. In the earlier stages of development it is very difficult to tell an adenoma from a cancer growing from the same part, for in each the epithelium is at first confined to the ducts and acini of the gland.

Adenoma has been observed in almost all the situations where gland tissue normally exists. We suspect that secondary epithelioma of the liver has been mistaken for adenoma. It is next to impossible to differentiate the two by the microscopical characters; and if there be only a single nodule the difficulty is increased, especially if its growth be slow, and it possess an adventitious capsule from irritative overgrowth of the hepatic interstitial tissue.

\* Vide *Trans. Path. Soc.*, vol. xxxiii. pp. 290 and 335.

## CHAPTER XCIX.

## CYSTS.

THE classification of cysts can be made on several bases : (1) Their origin ; (2) their contents ; (3) the organs and tissues affected. The first is followed in this work ; it is a modification of the system adopted by E. Wagner.

1. Cysts derived from the distension of natural cavities.

(a) Cysts that have their origin in closed spaces.

(a) *Serous cysts*—*e.g.* enlarged bursæ, ganglia on the sheaths of tendons, spinal and cerebral meningoceles, and vaginal and funicular hydroceles.

(β) Cysts formed by hypersecretion in closed follicles—*e.g.* some ovarian cysts and cystic goître.

(b) *Retention cysts*, from partial or complete obliteration of ducts or orifices of glands.

(a) Sebaceous or atheromatous cysts.

(β) *Mucous cysts*—*e.g.* ranula.

(γ) Those containing a more specific secretion—*e.g.* salivary cysts, dropsy of the gall-bladder, hydronephrosis, and renal cysts.

(c) Cysts arising from the dilatation of obstructed blood-vessels and lymphatics.

2. Parenchymatous cysts, or those of independent origin.

(a) Cysts derived from the dilatation and fusion of connective-tissue spaces, such as bursæ formed beneath the skin in club-foot and corns ; also congenital tumours of the neck—hydrocele of the neck.

- (b) Adventitious cysts whose walls are constituted by the condensation of the connective tissue of the part. They are formed around parasites, hydatid tumours, blood clots, etc.
  - (c) Cysts of congenital origin, chiefly found in the ovary. They are called dermoid cysts, since their contents are made up for the most part of cutaneous and epidermal elements, glands, hair, teeth, etc.
  - (d) Cystic new formations, very common in sarcoma, enchondroma, and other growths. (*Vide* Fig. 10.)
3. Extravasation cysts.

(a) From effusion of blood into pre-existing sacs (hæmatocele).

(β) From diffuse extravasation into solid tissues, especially the new growths ; and into the brain.

**Serous cysts.**—(1) *Enlarged bursæ* are usually of inflammatory origin. The term *hygroma*, which has been applied to them, is by some pathologists extended to all serous and mucous cysts, and to lymphatic nævi, but more frequently it is employed in a more restricted sense to indicate cysts of lymphatic origin, such as congenital cystic tumour of the neck and its congeners. The most important are those situated in front of the patella, on the inner side of the knee and over the ischial tuberosities (“weaver’s bottom”) and great trochanters. The walls, which are composed of fibrous tissue, are sometimes quite thin ; at others thick and rugged. The cavity may be a mere chink or as large as a double fist.

The inner surface of the cyst is lined by flat epithelium, though this often becomes destroyed. The contents are clear and serous or mucoid, or turbid from admixture with blood and the fatty débris



of inflammatory products. In one case I extracted half an ounce of cholesterine from a single enlarged pre-patellar bursa. Sometimes they contain loose bodies, resembling those found in ganglia and articular synovial membranes.

(2) *Ganglia* are simple and compound. They are most common about the wrists and ankles. The compound ganglia are irregular dilatations of the sheaths around groups of tendons—*e.g.* the flexors of the fingers. Simple ganglia are pouch-like dilatations that have become shut off from the synovial sheaths. The contents of ganglia are usually clear and viscid. The compound variety often contains loose bodies, some of which appear to be fibrinous exudations concentrically laminated, but the majority are hypertrophied buds or fringes detached from the cyst wall.

**Cysts connected with diseased joints. —**

Morrant Baker and others have drawn attention to the fact that large cysts are sometimes met with, especially in the popliteal space and calf of the leg, associated at some period of their history with disease of the neighbouring joint (page 324). In most cases affection of the articulation has been known to precede the development of the cyst; in others the swelling caused by the latter has been first noticed. These cysts may arise (1) from hernial protrusion of some part of the synovial membrane of the joint; *e.g.* that connected with the tendon of the popliteus muscle; (2) from a connection being established between a joint and an adjacent bursa, the fluid first accumulating in the joint, and then by its pressure opening up a communication with the bursa; (3) by distension of a bursa primarily continuous with the synovial sac of a joint; (4) by rupture of the distended capsule of a joint and escape of synovia; (5) by a simple filling of a closed bursa

in consequence of inflammation spreading from a joint. Whether or not developed in connection with a pre-existing bursa, it may or may not be possible to trace a direct anatomical connection with the joint, either by surface examination or by dissection. They may attain a large size. When they occupy the calf of the leg they may appear continuous with the knee-joint, or lie in close proximity to it, or an interval of some inches may exist between the cyst and the joint. These cysts have been observed in Charcot's disease, rheumatoid arthritis, and simple serous synovitis.

**Closed follicular hypersecretion cysts.**—

(1) Simple ovarian cysts, not rare in children. They arise from distension of Graafian follicles, and do not proliferate like the compound cysts of the ovary.

(2) *Cystic bronchocele*.—The glandular acini of the thyroid body first become choked with epithelium; this undergoes colloid softening, which extends to the trabeculae. The stroma is partly destroyed by atrophy from the pressure of the fluid within. Thyroid cysts are single, or multiple and loculated; they may be larger than an orange. Their contents are viscid and yellow, or thin and either clear or turbid. (*Vide* page 133.)

**Retention cysts.**—1. *Sebaceous cysts*.—This group includes molluscum contagiosum, which is really an adenoma of the skin, strophulous albidus, comedones, and the ordinary form of sebaceous cyst.

(a) *Molluscum contagiosum*.—This consists of small tumours looking like “drops of white wax” (T. Fox). They are seated for the most part on the face, neck, trunk, and arms. They vary from the size of a pin's head to that of a marble. They are umbilicated in the centre, the depression marking the site of the duct. They are sessile, or broadly pedunculated. The nature of the contagiousness is uncertain. Fox says it is not parasitic.

(b) *Comedones* are small hard elevations caused by distension of the sebaceous glands. Their apices present black specks from accumulation of dirt. When indurated from irritation we have *acne punctata*.

(c) *Strophulus albidus* of children is similar to the last. It has the appearance of millet grains. The obstruction of the gland orifice is due to the irritation of heat and moisture.

(d) *Sebaceous cysts proper*—*atheromatous cysts* are found chiefly in the scalp, but most parts of the skin are subject to them.

In the scalp they are rarely larger than a walnut, but in other parts (*e.g.* the back) they sometimes exceed the size of a large orange. There is usually obliteration of the glandular orifice. The skin becomes stretched over the tumour, and its distinctive structure modified by atrophy from pressure or hypertrophy from friction. As the cyst enlarges it occupies the subcutaneous tissue, and it may then be mistaken for chronic abscess or a soft fatty tumour. The cyst wall is made up of parallel layers of connective tissue. The contents may be soft like honey (*melicerous cysts*); fluid, when it is clear or chocolate or sepia-like from fat and blood pigment; or quite firm (*cholesteatoma*); under the microscope fat granules, epithelial cells, cholesterine, and fat crystals can be recognised in varying proportions. The epithelium lining the cyst is stratified. The cells next the cyst wall have large nuclei, and show signs of proliferation; farther in they are of the ordinary pavement variety, whilst those abutting on the cavity are filled with fat and devoid of nuclei. This epithelial lining is sometimes of horny consistence, the cells being welded together so that the entire lamina can be shelled out. The cysts sometimes rupture and give rise to inveterate ulcers that may be mistaken for epithelioma. This is due to the persistence of the

epithelium in the interior of the cysts; hence in operating upon the latter care should be taken to remove the whole of the cyst wall. Sebaceous cysts are by some spoken of as *wens*, a term also applied to a variety of fibroma (page 502).

Sebaceous cysts are occasionally found in the palm of the hand. They arise from portions of epidermis carried beneath the skin by injury, and are known as *implantation cysts*.

2. *Mucous cysts*.—These are met with in the lips and buccal mucous membrane. They are not uncommon in the floor of the mouth, where they constitute one form of *ranula* (others are derived from obstruction of the salivary ducts and from free cyst formation, and the contents vary accordingly). Labial and buccal mucous cysts look transparent, and contain a viscid secretion. Cysts of like nature are seen in the stomach, intestine, trachea, and uterus.

3. *Cysts containing specific secretion*.—Amongst these are *salivary cysts*, the cause of which is inflammation about the ducts, or impaction of a calculus. *Hepatic cysts* contain the constituents of bile. They arise in one of two ways, either from obstruction of the small biliary ducts, or thrombosis of the sublobular veins. Their contents are often putty-like.

*Renal cysts* involve the entire cortex, so that the organ looks something like a bunch of grapes; or they are isolated and scattered. The former, which may be congenital, are essential cystic formations; the latter are more or less accidental, and are found associated with other morbid conditions, especially gouty kidney; they are the result of obstruction of the tubules, and the wall of the cysts is usually a distended Malpighian capsule. These retention cysts may contain urates (Virchow) and urea (Cornil and Ranvier).

Congenital tumour of the kidney is commonly a cystic adenoma (page 466).

**Cysts arising from the dilatation of obstructed blood-vessels and lymphatics** are not very common. In the case of the blood-vessels the circulation is arrested, thrombosis follows, and the clot undergoes liquefaction. The wall of the vessel becomes much altered, and the cyst enlarges by the exudation of serum. They are met with in obliterated naevi.

Lymphatic cysts are lined by tessellated epithelium, and their contents clear from the first.

**Parenchymatous cysts.** — 1. *Bursæ* are developed where there is intermittent pressure. Embryonic is converted into mucous tissue, and the cells and fibres of the latter undergo mucoid liquefaction. The external cells remain as an endothelial lining to the cysts.

*Congenital cystic tumours of the neck* often attain a large size. The solid portion of the growth may predominate over the cystic; there is great variation in this respect. The cysts form in embryonic tissue, the cells of which become distended with mucin and then disappear. They may arise from dilatation of lymphatic spaces. *Hydrocele of the neck* is a serous cyst. It may be likened to the subcutaneous bursa, though no cause may be assigned for its development. I once saw one as large as an ostrich's egg; it was translucent, and was filled with limpid fluid; it was surrounded by loose areolar tissue, and the wall, which was very thin, had an exquisite mosaic of pavement epithelium on the inner surface.

2. *Adventitious cysts* require no further description than is given in the table.

3. *Dermoid cysts* are met with chiefly in the ovary. They are usually congenital. Their cavities are single or loculated. That they are not the outcome of so-called *fetal inclusions* is proved by the fact of their having occasionally been found to contain more than a hundred teeth. Tissues of epidermal origin



predominate, hair, horny epithelial buds, and teeth; but striated muscle, medullated nerve fibres, nerve-cells, and bone have all been seen. The hairs are usually shed in succession, so that large tufts or loosely woven balls are found free in the cavity of the cyst, embedded in desquamated epithelium and fatty débris.

The author examined a specimen, in the loculi of which were masses of fat like butter; the cyst wall was set with piliferous papillæ.

The ovary is the seat of election, because it is destined in the course of nature to produce cells that have the potentiality of developing into all the tissues of the body. These cysts are congenital, or occur early in life, when the developmental activity is great.

Their growth may extend over many years. On the other hand, they may make rapid strides after a lengthened period of quiescence. A patient under my observation died from what was thought to be encephaloid cancer filling the abdomen, but *post mortem* the growth turned out to be a dermoid cyst, which contained nothing but hair and pultaceous epithelial débris. They may become sarcomatous.

4. Cystic sarcoma, enchondroma, etc., are described under their respective headings.

*Proliferating ovarian cysts.*—The origin of these cysts is doubtful. It is more likely that the primary spaces result from mucoid or colloïd softening of aggregations of new formation cells than that they arise from distension of Graafian follicles. At first they are small, but they rapidly enlarge by absorption of the intervening walls, and by the addition of the softened products of cell proliferation. They become occupied by papillated growths that spring from the inner surface. Secondary cysts are formed by adhesion of the tips of contiguous papillæ (W. Fox), or by softening. The contents of compound ovarian cysts may be thin and serous, but more often the fluid is



quite viscid, consisting largely of mucin. Cells of various sizes and in different stages of fatty and mucoid degeneration are suspended in it. Occasionally it sparkles with crystals of cholesterine. It may be tinged or more deeply coloured from altered blood. Hæmatoidin crystals have been seen.

The cells next the cavity of the cysts are large and distended with mucin, or granular and fatty. Next come polygonal or rounded cells, whilst those immediately lining the wall are generally columnar. Only the last are seen when the mucoid transformation is complete, and then microscopical sections closely resemble adenoma or even columnar epithelioma.

**Extravasation cysts** are very common in soft malignant growths, such as encephaloid cancer and sarcoma. Of the natural tissues the brain is the most likely to be the seat of cysts as the consequence of hæmorrhage. This is explained by the physical disabilities which hinder the collapse of the brain tissue around blood clots in process of absorption—viz. the peculiarity of the intracranial circulation and the fixity of the cranial walls. They are common between the arachnoid and the dura mater.

**Hydatid cysts.**—Two forms of hydatid cyst occur in man—*echinococcus* and *cysticercus*. The former is the cystic stage of the tapeworm of the dog; the latter usually of the *tænia solium*.

**Cysticercus cellulosæ** is very rare in man, and never attains a great size. It has been observed in the humours of the eye, in the brain, and other parts. The animal is always solitary in its cyst.

**Echinococcus.**—On arriving in the intestines the ova lose their envelopes, and the embryos, set free, make their way into the different tissues of the body. Their transit is mainly by the blood-vessels, and by way of the common bile duct; hence the

frequency of hydatid cysts in the liver. At last they settle down and become encysted. An adventitious fibrous capsule forms around the essential cyst. Secondary and tertiary cysts develop within the primary. The vesicles vary from the size of a pea to that of an orange. Each is composed of a number of

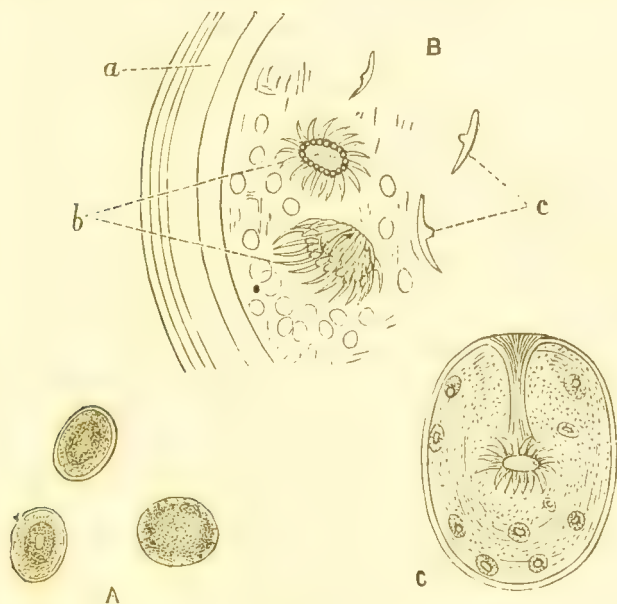


Fig. 95.

A, Ova of *tania solium*; B, portion of fertile cyst showing, a, laminated membrane, b, echinococci attached to germinal layer, c, detached hooklets; c, echinococcus; the head and neck are withdrawn within the caudal vesicle.  $\times 265$ .

superposed parallel laminae of perfectly homogeneous material, an appearance quite characteristic (Fig. 95, B, a). The innermost lamina is called the germinal membrane, and from it the echinococci develop. The latter look like white specks to the unaided eye. Each consists of a head and neck, which are usually retracted within the caudal vesicle (Fig. 95, c). The

head is surmounted by a proboscis; it carries four suckers or vents, and two circlets of spines.

Unless degenerated, the cysts are transparent. They give a peculiar trembling sensation to the hand (hydatid thrill). They are filled with clear fluid, which contains a little common salt, but no albumin. In the fluid detached echinococci, isolated hooklets, and shreds of disintegrated laminae may be seen at times. I once removed a hydatid tumour from the thigh of a woman; it had been growing for seven years, and was larger than a fetal head. Most, if not all, of the cysts were barren (acephalocysts).

The disease known as "sturdy" or "gid" in cattle and sheep is due to hydatid of the brain. The parasite is the cystic stage of the *tania caninus* of the dog. It is beautifully mottled with pigmented corpuscles.

There are four characteristic diagnostic signs of hydatid cysts: (1) The laminated membrane; (2) the fluid; (3) echinococci; (4) hooklets. To seek the last two, the fluid should be allowed to stand, and the lower part drawn off with a pipette, and submitted to the microscope. As hydatid cysts enlarge, the continuous pressure causes atrophy of the surrounding tissues.

Withdrawal of the fluid generally suffices to kill the parasite and put a stop to further growth of the cyst.

They are found in all parts of the body, even in the bones.

**Secondary changes.** — (1) The adventitious cyst may calcify, and be converted into a kind of carapace.

(2) Death of the parasite, and shrinking and folding up (Fig. 7), or disintegration of the cyst walls. The hooklets are set free on breaking up of the echinococci; they may be found in old obsolescent cysts.

(3) Fatty degeneration and caseation of the walls of the cysts.

(4) Importation of foreign matter; bile, blood-pigment, etc.

**Modes of termination.**—(1) Obsolescence; (2) suppuration around the cyst; (3) rupture, with its consequences: (*a*) diffuse inflammation (peritonitis, pleurisy); (*b*) hæmorrhage.

## CHAPTER C.

## THE CARCINOMATA.

TYPE: epithelium. There are two forms, commencing respectively in glandular and surface epithelium, but their clinical and pathological affinities are strongly marked; thus both generalise in the lymphatic glands, and almost always end fatally.

As to the **cause of cancer**, two agencies are at work, local and constitutional. It would appear that each may be sufficient in itself, but there can be no doubt but that in the majority of cases they act together, though frequently in inverse ratio.

In support of the *local origin*, (1) we may mention the cases where continued or repeated irritation is followed by a cancerous growth—*e.g.* epithelioma of the lip and tongue from the friction against a pipe or tooth, and “chimney-sweep’s cancer” of the scrotum; (2) taking the alimentary canal, it is at the places most subject to friction that these tumours are found; lip, tongue, fauces, upper and lower ends of œsophagus, and, where the left bronchus crosses it, pylorus, ileo-cæcal valve, rectum, and anus; (3) organs that are liable to vicissitudes of function, as the mamma in the female.

In favour of the *constitutional origin* are (1) those instances where no unusual irritation can be discovered; (2) those where the primary outbreak is multiple; and (3) those where hereditary predisposition is strongly marked.

No age is exempt, but it is much more common at and beyond mid-life. The disease is characterised by a continuous non-inflammatory local growth, usually repeated in distant parts, and by an apyrexial wasting

of the whole body, with a sallow, greenish yellow, or leaden hue of the skin (cachexia).

**Generalisation** takes place: (1) In the glands through the lymphatics, and this explains recurrence after removal, in the tissue intervening between the primary and secondary growth. (2) By the blood-vessels in the internal organs and other parts. (3) It may be that where a nodule projects from the surface of a serous membrane, particles may be broken off and form new centres of growth.

The tumour grows (1) by multiplication of its own elements; (2) by a "spermatic influence" upon indifferent cells in contact with it (Cornil and Ranvier); (3) by discontinuous tubercles, which, at first isolated, join one another and the main growth as they enlarge. They are probably connected by the lymphatics from the first.

**Alveolar or glandular cancer**, of which scirrhus is the type, shows a well-defined structure, in which the cells of epithelial origin are packed in regular spaces or alveoli, bounded by trabeculae of fibrous tissue, containing small embryonic corpuscles. Blood-vessels and lymphatics are found in the stroma, but only the latter enter the alveoli. There are probably no intrinsic nerves, those seen in sections belonging to the tissue invaded by the growth. The cells filling the alveoli are large, polymorphous (round, angular by pressure, tailed, etc.), and contain bright nucleolated nuclei. Anatomically, there is no such thing as a recognisable "cancer cell"; physiologically, no doubt, each one embodies in itself all the attributes of the disease. The stroma varies greatly in amount, according to the rate of growth of the tumour; it is usually fibrous, but when recent it often appears like spun glass, or it is homogeneous.

**Scirrhus cancer of the breast** is very hard, and cannot be enucleated from the tissue in which it



grows. It is often more or less lobulated on the surface. On section, a well-marked cupping is observed, for the older central part is denser and more contractile than the peripheral. To the naked eye it appears like an unripe pear, having a greyish semi-translucent look, varied by yellow streaks and specks, which mark alveoli and obstructed milk ducts filled with the fatty debris of degenerated cells. The growing marginal zone is faintly pink, for here as yet the vessels are intact.

Not seldom small cavities are to be seen, the result of obstruction of lymphatics and ducts; these contain a clear pale fluid, or this variously stained by the colouring matter of the blood. I have shown that as a rule when old they are lined by tessellated epithelium. During life a clear or sanious fluid sometimes oozes from the nipple or can be expressed from it. The fibrous stroma, as it shrinks, obliterates most of the vessels; this causes degeneration and wasting of the cells, so that alveoli become mere chinks filled with shrunken cells and granular debris, and finally disappear, a coarse-grained cicatricial tissue being all that remains. Meanwhile the tumour increases at the periphery, and destroys the tissue it invades and infiltrates. Other things being equal, the rate of growth varies inversely, and the contraction directly, as the age of the patient. In old women, after the lapse of several years, what remains (if any) of the diseased gland, together with the tumour, may be of less bulk than the healthy breast. This is termed *atrophic scirrhus* (Fig. 96, B).

In its march scirrhus cancer attacks all the structures in its course, so that it becomes adherent to the skin over it and to the pectoral muscle, and, it may be, the ribs and pleura beneath. The cicatricial contraction accounts for the wrinkling and puckering of the skin, and for the retraction of the nipple.

If left to itself the skin usually becomes inflamed and then ulcerated, and the cancerous tissue covered by a layer of granulations is deeply excavated ; or it forms a protuberant fungus. In some cases nodules are seen in the skin around the central growth ; in fact, the skin may bear the brunt of the mischief, being thickly studded with tubercles that, coalescing, constitute the cancer *en cuirasse*, hide-bound cancer, shield cancer, squirrhe pustuleux (Velpeau).

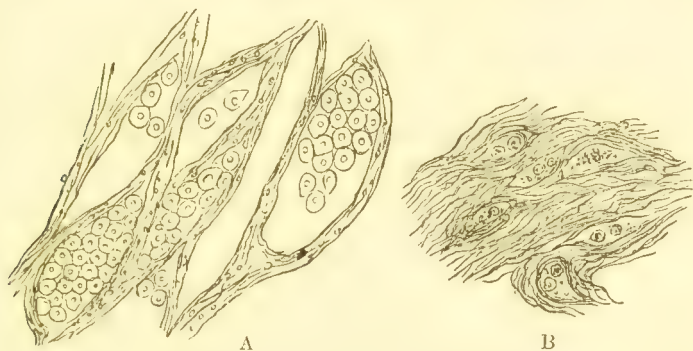


Fig. 96.—Scirrhus Cancer of the Breast.

A, Rapidly growing form ; B, atrophied or cicatricial scirrhus. Both show alveolar structure. In A the stroma is scanty and the epithelial cells distinct. In B the stroma is dense, the alveoli shrunken, and the contents in a state of degeneration.  $\times 265$ .

The more rapid the growth the more likely are the axillary, mediastinal and supraclavicular glands to be affected. In atrophic scirrhus they escape for a long time, but eventually they are involved, with dissemination in the internal organs. The male breast is the seat of the disease in about 2 per cent. of the cases (Paget).

Cancer of the breast is sometimes “acinous” ; that is, the original outline of the gland acini is more or less preserved ; this accords with the lobulated outline of these growths. But there is a “tubular” variety, in which the cancer cells permeate the gland

tissue in narrow columns; the difference seems to depend upon the point of departure of the new epithelial formation; in the latter instance (tubular) it probably starts in a hyperplasia of the cells lining the ducts.

An *eczematous condition of the nipple* (Paget's disease) may precede the development of cancer in the gland by months or years. The obstinacy of the eruption and its circumscribed margin show that it is not a simple eczema, and against its being primarily cancerous is its long duration; but it may be that ordinary glandular cancer in some cases commences in a slow derangement of nutrition of the epithelium, not thought of because not seen, and that it is only later that it manifests itself in an open "rebellion of cells." Analogous to the above is epithelioma, as a sequel to "psoriasis" of the tongue.

This affection and the allied disorder of the scrotum associated with "chimney-sweep's cancer" has been referred to the agency of organisms termed *psorosperms*; but their causative relation to the disease, even if they exist, is doubtful.

*Microscopy.*—To understand the minute anatomy of scirrhus, sections should be made of different tumours and different parts of each. In a rapidly-growing scirrhus the *stroma* at the periphery is imperfectly fibrillated, sometimes quite homogeneous, but in the older portions the fibrous trabeculae are very distinct. There is a marked relative increase as the centre of the growth is approached, for as the cicatricial contraction obliterates the vessels, the cells atrophy and the stroma shrinks, so that the alveoli get smaller and smaller. The more rapid and recent the growth the more is the stroma infiltrated with embryonic cells, which are sometimes so numerous as to hide the matrix. The *alveoli* vary in size and shape from the first, but are for the most part fusiform, and

double the width of the trabeculae bounding them. They are not closed spaces, as they at first sight appear, for they constitute a system of intersecting canals, which can be seen in thick sections by altering the focus of the microscope. As the stroma shrinks they necessarily diminish in size. The *cells* filling the alveoli are variously-shaped—round, angular, caudate, etc., according to the extent of the pressure upon them. Their size is in marked contrast with the indifferent corpuscles of the stroma.

Except at the periphery, the *blood-vessels* can only be seen in injected specimens. They become thrombosed through the contraction of the stroma. In the oldest portions of the growth they disappear, from atrophy of their walls. It is said that by the aid of nitrate of silver the *lymphatics* can be traced in fresh specimens into the alveoli.

Any fat cells met with in the tumour belong to the tissue invaded. The epithelial cells of cancer never become distended with fat drops. In this way they differ from some cases (page 547) of sarcoma (Cornil and Ranvier).

**Encephaloid cancers** have exactly the same structural arrangements as scirrhus, but differ from the latter in the relative amount of stroma and cells. They are usually soft, brain-like tumours, though sometimes they are moderately firm in consistence. The alveoli are larger than in scirrhus, and the trabeculae for the most part more delicate. Moreover, their tendency is to degenerate and soften; in fact, they may be diffuent in the older portions, or even throughout; hence hæmorrhages into their substance are common. When the surface becomes ulcerated the unrestrained growth protrudes in the form of "*fungus hæmatodes*" (page 408). They are so elastic that they often give a sense of fluctuation. In colour they are yellowish-white, mottled, or flecked with red; here and

there may be seen patches of modena hue, from capillary extravasations. Blood-cysts are not uncommon. Usually of rapid growth, and very malignant, there is, nevertheless, a wide range in morbid appearances and clinical course.

In St. Mary's museum is a specimen of encephaloid cancer of the testicle removed during life; it is

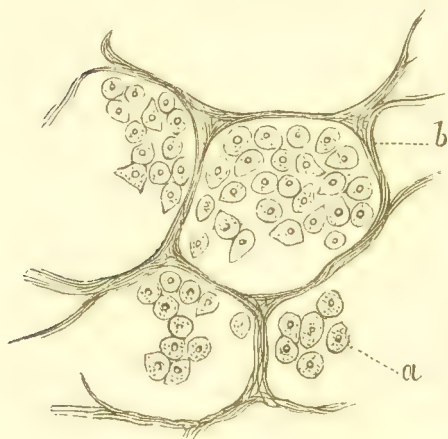


Fig. 97.—Encephaloid Cancer of the Testicle.

*a*, Epithelial cells; *b*, fibrous stroma.  $\times 265$ .

firm and fibrous in one half its bulk, pultaceous in the other. The tumour, as large as a foetal head, had been growing for four years without any signs of thickening of the cord, secondary growth, or constitutional taint.

Both scirrhus and encephaloid yield a milky juice on scraping.

**Microscopy.**—The alveoli are large, and round or oval in shape, rarely narrow and fusiform as in scirrhus. The *stroma* is subject to great variation both in nature and extent. It generally forms delicate overarching bands (Fig. 97, *b*), but at times wide dense trabeculae can be seen; but even then we do not find the shrunken alveoli so constant in scirrhus. It may be entirely fibrillated, or consist of a few fine fibres traversing a homogeneous matrix. It may be almost devoid of indifferent cells, or thickly strewn with them. The *epithelial cells* filling the alveoli are large. They are not so multiform as in scirrhus, for the intercellular substance is less dense and more



copious; and hence the cells are not so liable to alterations in shape from pressure. They contain large nuclei, one or more in each cell, with bright, highly refractive nucleoli. When the cells are undergoing degeneration fat particles make their appearance in the protoplasm. The *vessels* are wide, and have very thin walls, and from defective and unequal support they develop ampullary dilatations. These frequently rupture, and give rise to extravasation of blood.

**Colloid cancer** is built upon the same structural type as scirrhus and encephaloid. It bears a close resemblance to the latter in its clinical features, being rapid in growth, and quickly fatal. It selects for its ravages many different tissues, but the bulk of the cases are met with in the *abdominal viscera*, especially the intestinal tract and the ovaries. The peritoneum is often seen to be infiltrated; but on the assumption that all cancers start from pre-existing epithelium, it must be granted that these omental and mesenteric growths begin in the stomach, intestine, pancreas, or liver, since the epithelioid lining of the peritoneum belongs to the connective-tissue series. The open meshwork of the serous membrane is a favourable ground for the spread of the disease, richly provided as it is with blood-vessels and lymphatics.

The consistence of these tumours is subject to wide variation, but for the most part they are very soft, sometimes diffuent. When springing from the ovary they may be mistaken for simple cystic formations.

Cancer is rare in the *thyroid body*; but when it occurs, colloid matter is seldom absent. In this way it conforms to the rule that most of the morbid changes found in the gland are accompanied by colloid degeneration. The alveoli are larger and less angular than in scirrhus, for the colloid transformation gives rise to an increase in bulk, and this expands the walls of the



spaces, and renders the latter globular or ovoid. The degeneration commences in the cells. First a drop appears in the protoplasm, and as it enlarges, the nucleus is thrust to the margin. Finally, nuclei and cell capsules disappear. The change advances from the centre to the periphery, and the outside cells, prior to their destruction, become compressed and

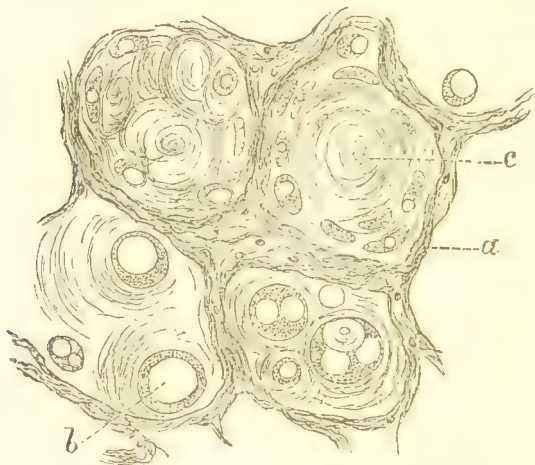


Fig. 98.—Colloid Cancer of the Ovary.

*a*, Fibrous stroma; *b*, cell distended with drop of colloid material; *c*, alveolus. The cells that occupied the centre have disappeared, and in their place a semi-homogeneous mass marked by concentric streaks can be seen. The peripheral cells are flattened from the pressure of the colloid substance.  $\times 265$ .

elongated, and occupy a concentric position (Fig. 98). The stroma undergoes a similar alteration; it softens and liquefies, so that contiguous alveoli run together, forming festooned cavities.

These unfractuous spaces, bounded by tracts of fibrous tissue, give an alveolar disposition that can be seen by the naked eye. The secondary growths are of the same nature as the primary.

**Columnar epithelioma.**—This variety affects the stomach and intestines (especially the rectum), the

bladder, ovary, cervix uteri, nasal fossæ, and the jaws. The pattern is exquisitely alveolar, like a piece of mosaic. The *stroma* is now scanty and delicate, and again excessive and coarsely fibrillated, the variation being often marked in the same specimen. In some parts it is almost free from corpuscles; in others crowded with small round, indifferent cells. The epithelial cells line the acini with great regularity. They are large, columnar, and have bright elongated nuclei (Figs. 10, 75). Sometimes they become distended with mucin, and present an inflated appearance. The tumour, when it reaches the surface, quickly ulcerates, and, being very vascular, bleeds profusely, the more so when subjected to mechanical irritation; *e.g.* by the passage of fæces over it.

It has a less tendency than scirrhus or encephaloid to generalise in the lymphatic glands. In the case of the *rectum* secondary growths may be disseminated in the liver. The path of infection would seem to be along the branches of the portal system. These secondary growths in the liver possess the same disposition and shape of the epithelial cells as the primary lesion.

I have observed a columnar epithelioma of the bladder in which the alveoli were separated by a very scanty stroma, and the cells, arranged in superposed columns, completely filling the spaces.

In the *jaws*, particularly the lower, the central cells are round, or angular by compression, and the peripheral columnar. In this situation the new formation is almost invariably cystic, and it is noteworthy that only the central cells undergo mucoid degeneration, the columnar ones at the periphery remaining as an epithelial lining to the cysts (Fig. 10). Here, too, the interacinose tissue is fibrous, or composed of spindle-shaped cells; in the latter case the growth has for this reason been mistaken for sarcoma.

Cystic epithelioma of the jaw is slow in its progress, and has but little tendency to involve either the lymphatic glands or the internal organs.

It commonly starts as an epithelial proliferation of persistent portions of the enamel organ of a tooth (*vide* Odontomata, page 520).

**Squamous epithelioma**, also called epithelial cancer, in contradistinction to scirrhus and encephaloid, which were formerly believed to be connective-tissue tumours, but since all cancers start from pre-existing epithelium this differentiation is groundless. They are found for the most part in situations that admit of their operative treatment, and they illustrate the principle that local irritation is a noteworthy factor in the aetiology of malignant growths, whether this be in the form of a precedent inflammatory lesion—*e.g.* syphilitic ulcer of the tongue—or mechanical friction, as from a sharp tooth or the stem of a pipe. Their tendency to develop at the junction of skin with mucous membrane is well known; but this is explained rather by the coincidence that these are the very places most exposed to irritation than by any inherent disposition of the epithelium to perverted growth. That the surface epithelium preponderates in the lips *e.g.* as the age advances, is no proof that its nutritive activity is greater. The increase is relative rather than absolute, for in old people the connective tissue and muscular fibres waste. The favourite situations where skin and mucous membranes meet are the lips, genital organs, and anus. The general cutaneous surface is by no means exempt; the face and back of the hand take the lead, if we exclude the skin of the leg, where some simple chronic ulcers at length become cancerous.

The mucous membranes, naturally covered with laminated scaly epithelium (*e.g.* the tongue, cheek, fauces, œsophagus, vagina, and cervix uteri), are all

liable to the inroads of the disease. In the bladder the deep epithelial cells are columnar, and the superficial flattened; hence in this viscus both squamous and columnar epithelioma are met with. Epithelioma of the scrotum is known as chimney-sweep's cancer, it being thought that the friction of the sharp particles of soot keeps up a chronic irritation; and some weight is given to this view by the fact that the disease is less common than formerly, when chimneys were swept by climbing.

The first effect of the local irritation is to cause a simple inflammatory hyperplasia, and this may go on for a long time before any specific epithelial overgrowth takes place. Eventually this happens, and the multiplication of cells proceeds from the deeper layers of the epidermis and its involutions—the hair-follicles, sebaceous and sweat glands. In some cases this is mainly confined to the surface—in others it infiltrates the structures beneath. There is great variety in this respect.

Rod-like masses of epithelium advance in the direction of least resistance—*i.e.* between the bundles of connective tissue, it is said, in the lymphatic spaces. If this be so, we have a ready explanation of infection of the lymphatic glands next in order to the primary growth. These epithelial cylinders send off buds in various directions, which increase at the periphery, and thus form the well-known “epidermal globes” or “epithelial pearls.” Concentric lamination of the cells explains the characteristic “bird’s nest” appearance on section (Fig. 99). This lamination is the result of pressure, aided by the shrinking of the older epithelial cells as they become horny; the latter process is a repetition of what takes place physiologically in the superficial layers of the epidermis. The “globes” are not all isolated groups of cells, but many are the transverse sections of the down-growing

columns above referred to. Some are microscopical, but others can be seen by the naked eye as yellowish spots embedded in the indifferent growth that surrounds them. As the epithelial cylinders increase in size they project from the surface, giving it a granular

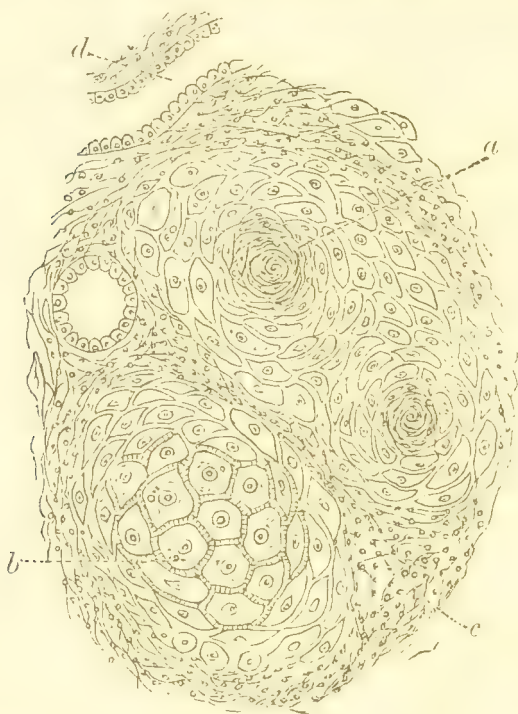


Fig. 99.—From an Epithelioma of the Thumb.

*a*, Epithelium "nest"; *b*, large polygonal cells marked by fine striations at their borders; *c*, indifferent tissue; *d*, sweat duct.  $\times 265$ .

appearance. By pressure they can be squeezed out like the contents of a sebaceous gland. The peculiar hardness of this form of epithelioma (useful in diagnosis) is due to the cornification of the older cells in the same way that the epidermis, with its appendages, the hairs and nails, becomes horny.



Now picro-carminic with an excess of picric acid stains the rete mucosum red, and the cuticle bright yellow; and as the same chemical changes occur in the cell-columns of epithelioma as is found in the normal evolution of epidermis, and as these columns increase in width mainly by the addition of new cells to the periphery, it follows that the central cells of the epithelial nests should be stained yellow, and the outer ones red, and so it is found in the greater number of cases. In some, however, the central cells continue to enlarge and multiply, whilst the peripheral ones become compressed; the result is a collection of large spherical, polygonal, or oval cells, surrounded by laminae of flattened ones.

The connective tissue and other structures invaded by the proliferating epithelium show an irritative hyperplasia. Indifferent cells crowd the stroma. The old blood-vessels dilate and new ones are formed. The inflammatory new formation leads to softening and ulceration. The destruction progresses from without in. In some cases the growth has but little tendency to spread deeply, but gives rise to large-branched projections termed *cauliflower excrescences*. The blood-vessels being less compressed than in the infiltrating variety, the necrosis of tissue is not so great; in fact, at first sight it is difficult to say whether the tumour be a papilloma or a papillary epithelioma. Closer examination reveals some invasion of the tissues beneath the base of attachment, and perhaps also lymphatic enlargement. A parallel case is furnished by the simple villous tumour and villous cancer of the bladder. However great the ulceration and sloughing may be, the constructive process is always greater than the destructive; the epithelial and inflammatory formation is heaped around the ragged margins of a deeply excavated ulcer, and projects in nodules from the indurated base.



There is great variation in the degree of malignancy. Some cases recur rapidly after removal, infect the lymphatic glands early, break down into gangrenous sores with foul discharge, and end fatally within twelve months. Others take years to run their course, and some are completely eradicated by operation. The more vascular the tissue affected the graver is the prognosis. In epithelioma of the tongue and mouth the general emaciation and discomfort are increased by the pain and difficulty in swallowing, and matters are made worse by the passage of decomposing discharges into the stomach and lungs.

Capillary and even arterial bleeding from ulcerated epithelioma is a great source of distress. It is often difficult to stop, and it recurs after short intervals, for the blood-vessels cannot contract, and fresh ones are opened by ulceration and sloughing.

The secondary deposits in the lymphatic glands have the same histological characters as the primary growth. Infection of the internal organs is rarely seen.

**Microscopy.**—If a vertical section be made through the margin and base of an ulcerated cutaneous epithelioma, it will be observed that the cells pass in columns into the subjacent structures. These columns follow the course of the hair follicles, sweat ducts, and sebaceous glands. They are not, however, confined to them, but ramify in the interstices of connective tissue. Since the buddings from the columns lie in different planes many are divided transversely and obliquely, giving one the idea of isolated cell masses. The characteristic arrangement in concentric laminae has already been alluded to as the result of lateral pressure and shrinking of the older cells from cornification. The flattening of the cells is in parts so complete that they look like fibres. Their real nature is manifest in the gradations of size and shape, from the centre to

the periphery of the "epithelial globes." Moreover, they can be isolated by the aid of liquor potassæ and teasing. The epithelial cells adhere firmly to one another either directly or through the medium of a scanty intercellular substance.

Many appear to interlock by fine serrations at their margins (stachel and riff cells) (Fig. 99, *b*).

The epithelial masses are surrounded by connective tissue studded with indifferent cells.

In some cases it is very scanty ; in others it exceeds in amount the epithelial portion of the growth. The ground substance appears homogeneous, or more or less perfectly fibrillated. The extent of the small-celled infiltration varies much in different cases ; generally speaking, it may be said to be directly proportionate to the rapidity of growth and the tendency to ulceration, and so to the local malignancy of the disease.

The epithelial columns enlarge by segmentation and endogenous formation of their own cells, and, it is said, by the conversion and appropriation of indifferent cells at the margin (Cornil and Ranvier), but we cannot subscribe to the latter proposition.

Blood-vessels ramify in the stroma, but do not enter the epithelial masses, and may therefore be said to be extravascular.

The stroma is less distinctly alveolate than in the other forms of cancer.

The individual cells are very large, averaging  $\frac{1}{700}$  inch in diameter. They do not show such diversity of outline as in scirrhus and encephaloid. Cornification of the cells is characteristic of this form of cancer. The intercellular substance is much more scanty than in scirrhus cancer.

*Secondary changes in epithelioma.*—Inflammation and ulceration are constant. Fatty, mucoid, and pigmentary degeneration of the epithelial cells is comparatively rare.

**Rodent ulcer** may be conveniently discussed here. Formerly it was considered to be distinct from epithelioma in its structure and clinical course. Paget says "it does not contain epidermal globes, nor any other elements of a cancerous nature"; but later observations have shown that this view is not correct, and that too great a similarity exists between rodent ulcer and the more chronic forms of epithelioma to justify its being regarded as other than "the least expressed form of malignant disease" (T. Fox). In short, it is a variety of epithelioma.

Rarely beginning before the fiftieth year, it pursues a steady and certain course, so slow that after certain years it may not exceed the size of a florin. The lymphatic glands may become enlarged from simple irritation, and even suppurate (Moore), but they are little liable to specific deposit by infection from the primary growth. In its ravages rodent ulcer spares no tissue, not even bone. Disseminated nodules around the primary lesion are generally absent. The older central part of the ulcer may dry up, but there is little or no approach to cicatricial contraction. The face is the favourite locality, though the disease is met with in other situations. The cellular elements composing the walls and base are for the most part the same as in simple chronic ulcers, but there is in addition a decided epithelial proliferation. It is true the cells are smaller than in ordinary epithelioma, but like them they show a disposition, remote though it be, to concentric lamination. The new formation commences as a hyperplasia of the epithelial cells of the sebaceous and sweat glands. The rete Malpighii becomes atrophied by the pressure of the growth beneath, but it manifests little tendency to active change; this is in marked contrast to the part it plays in the common variety of epithelioma.

Extensive local destruction of tissue is compatible with the most perfect general health.

**Cancers contrasted with sarcomas.**—(1) According to Waldeyer, Billroth, and English pathologists, *all* cancers are developed from pre-existing epithelium. There is no difference of opinion as to the origin of epithelioma, but the glandular form (scirrhous and encephaloid) is asserted by Cornil and Ranvier to commence in a proliferation of the connective-tissue cells. They say that if Prussian blue be injected into the tumour, it will pass along a continuous path formed by cancerous alveoli and lymphatic vessels, and that the same arrangement will be observed in fresh sections stained with nitrate of silver, the endothelial connective-tissue lining of the lymphatics passing directly into the groups of cells filling the alveoli. In this way they account for the constancy of generalisation in the glands first in order from the primary growth; but it is one thing to show an anatomical connection between the cancerous elements and the lymphatics and another to prove that one springs from the other. Besides, epithelioma, which begins in a proliferation of epithelium, also propagates itself in the next absorbent glands.

Dr. Thin has shown that beneath the basement membrane of duct cancer there is a delicate feltwork of interlacing elastic fibres similar to that described by Henle around the milk ducts of the breast.

This anatomical likeness between the healthy gland tissue and the carcinomatous structure lends weight to the assumption that scirrhus of the breast is essentially an epithelial growth. We are fully convinced that all cancers are epithelial in origin.

Sarcoma has its type in embryonic connective tissue.

(2) *Arrangement of constituent elements.*—In glandular cancer this is very definite, the cells being

contained in spaces which intersect in all directions, like a labyrinth, forming a cavernous structure. The stroma bounding these spaces consists of fibrous tissue, in which the blood-vessels are distributed.

In sarcoma the cells are embedded in a ground substance, homogeneous or fibrous. The bundles of fibres, when present, follow the course of the vessels, and do not arch over and construct alveoli. The vessels may be considered as one great system of capillaries, whose walls are formed of the cells of the tumour itself.

(3) *Mode of generalisation.*—Cancers disseminate chiefly by the lymphatics, as might be inferred from the close anatomical connection between them. The infecting material of sarcomata is conveyed, in the majority of cases, directly by the blood-vessels, and the secondary growths are usually found in the internal organs. Even when the glands are affected, it may sometimes be through the blood-vessels, for the intermediate lymphatics are not always infiltrated. Moreover, the glands may be attacked by a continuous invasion from the primary growth, and not specially through the lymph channels. And, again, the initial cause of the disease in some cases selects the lymphatic glands for its ravages, so that it is not a question of dissemination at all.

(4) *Encapsulation.*—When sarcomata spring from fasciæ, the sheaths of muscles, and some other structures, they often present a well-defined capsule, and can be shelled out of their bed, except at their base of attachment, and the slower the growth the more easily can this be done. Cancers, on the other hand, infiltrate the surrounding tissue, showing a complete absence of a capsule, unless it be by chance derived from the fibrous tunic of an organ destroyed by the growth—*e.g.* the testicle. In many cases sarcomata appear as indefinite in outline as cancers. This is well seen in the bones and in the internal organs, so



too much stress must not be laid on this difference between the two groups of tumours.

(5) Taking them altogether, it may be affirmed that sarcomata are less liable to *recur after removal*; but the more malignant vie with cancers in this respect, whilst in the extent and the early period of their *generalisation* they sometimes outstrip them.

(6) *Cachexia*.—It is by no means rare to find a sarcoma of enormous size, the patient being in good general health; whereas the constitutional signs of cancer are generally well marked, and often at an early stage—so much so that a cachectic appearance is a valuable aid to diagnosis. When, however, the generalisation of sarcoma is extensive, and still more when the primary growth has ulcerated, the wasting of the body and sallowness of the skin are very manifest.

(7) *Age of the patient*.—Sarcomata are prone to occur earlier in life than cancers, which seems only natural, considering they are formed upon the type of embryonic tissue, which is indifferent in structure, and possessed of great nutritive and formative activity. This difference only holds good within certain limits, for the majority of cases of malignant disease of the testis, both cancerous and sarcomatous, occur between the ages of thirty and forty-five (Butlin); but even here there is a greater liability to sarcomatous than to cancerous growth during the first decade of life.

(8) *Nutritive changes*.—Sarcomata, consisting as they do of unstable developmental tissue, are liable to greater variation from secondary changes than cancers, whether it be towards a higher phase of evolution, as in the tendency to form cartilage or bone, or to a decline in vital activity, as in calcification and the formation of cysts from mucoid softening; or to a perversion of normal nutrition, as when they become



melanotic in the tissues of organs that are naturally devoid of pigment.

(9) When quite fresh, sarcomata do not yield a *lactescent juice* on scraping, but soon after removal a liquefaction of the intercellular substance takes place, and the cells are thus loosened from one another. Then a milky fluid can be easily obtained, certainly within twelve hours.

Speaking broadly, sarcomata are not so malignant as cancers; but they differ from them less in this respect than do the various forms of sarcoma amongst themselves; *e.g.* a round-celled subperiosteal sarcoma of a long bone may end fatally, with all the signs of malignancy, within six months; whilst a central myeloid, after amputation of a limb, may neither recur locally nor in distant parts.

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